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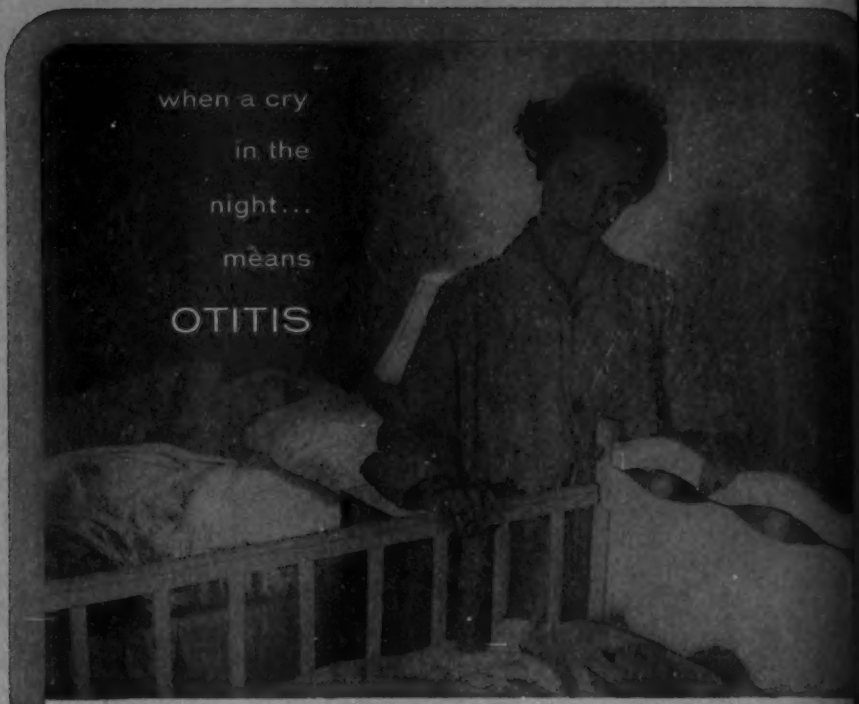

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1. Lazar, A. M., and Goldin, M.: Eye, Ear, Nose, & Throat Monthly 33:351, 1954. 2. Lazar, A. M., and Goldin, M.: Antibiotics Annual 1954/5, p. 468.

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THE
LARYNGOSCOPE.

VOL. LXVI OCTOBER, 1956 No. 10

SUDDEN DEAFNESS OF OBSCURE ORIGIN.*

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Sudden unilateral or bilateral impairment of hearing is a symptom, not a disease. The deafness under consideration in this report is sudden deafness of obscure cause—not deafness due to trauma, suppurative labyrinthitis, meningitis or mumps. The deafness is alarming to the patient and is almost always associated with roaring tinnitus. True vertigo may or may not be associated with the sudden deafness. If it does occur, it may precede the deafness, come on simultaneously, or come later. Such deafness is seldom or never associated with fatal events or diseases; therefore, factual information about the pathologic process underlying the loss of function is still obscure.

The physician who is dealing with deafness of this type must take the history carefully in order to decide whether the deafness came on suddenly. Fowler, Sr.,[1] stated that some persons have been known by their friends and relatives to be totally deaf in one ear for years but have not been aware of it themselves. The history may reveal that they suddenly noticed their deafness when the normal ear was buried in a pillow, and they could not hear a speaker standing a short distance away. Deafness also may and does actually come on like "a bolt from the sky"; sometimes the patient wakes up with it. In cases of sudden onset tinnitus is always severe and often distresses the patient more than the deafness.

* Submitted as Candidate's Thesis to American Laryngological, Rhinological and Otological Society, Inc., 1955.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication, January 20, 1956.

REVIEW OF LITERATURE.

Sudden deafness of obscure origin has received rather scant attention in the literature. Prosper Ménière[2] apparently was the first to report such a case and elaborate on the possible etiology and pathology. Kobrak[3] reported eight cases in 1922 but actually only one of his patients had sudden deafness with no obvious cause. De Kleyn,[4] in 1944, described "complete or partial loss of function of the octavus system in apparently normal persons." In the summary he mentioned 21 cases; actually he had 24 cases, in two of which the involvement was bilateral. He considered vascular disorders as the underlying cause. In some cases a more or less complete recovery was obtained. Rasmussen,[5] in 1949, reported 18 cases of sudden deafness of unknown etiology, in 15 of which the deafness was combined with vestibular disturbances. In discussing the etiology, he stated that in three cases the cause might have been thrombosis of the internal auditory artery or one of its branches, or hemorrhage into the labyrinth due to rupture of a vessel. In a fourth case vascular spasm was considered a possibility. In the remaining cases the cause was supposed to be neuritis of the VIIIth cranial nerve.

In March, 1950, Lindsay and Zuidema[6] gave an excellent review of 16 cases of inner-ear deafness of sudden onset. The first four cases were types in which the aural condition was associated with systemic disease. In the 12 cases in which the deafness was associated with a varying degree of vestibular disturbance the cause was uncertain. Lindsay and Zuidema further stated that the same etiologic factors cannot be assumed in all 12 cases, but that the similarity of the clinical picture suggests the same type of cause. A vascular accident in the labyrinth has commonly been the explanation, but the high incidence among adults less than 30 years of age argues against this cause. They also considered the possibility of vasomotor disturbances of the inner ear and recommended that toxic involvement of the labryinth be given serious consideration, since it is a well-known occurrence in infections of various types.

In December, 1950, Fowler[1] reported 26 cases of sudden deafness, and he attempted to include everything which might

have some bearing on the etiology. About 77 per cent of his patients were less than 40 years of age. Fowler's most important finding was a history of emotional episodes before or immediately preceding the sudden deafness. The patients, however, rarely seemed to blame the emotional condition for the deafness. Eighteen of the 26 patients apparently suffered from psychosomatic disorders. Fowler stated that such episodes are known to affect the neurovascular mechanisms through vascular contraction, sludging and congestion ending in anoxia and leakage from capillaries and local metabolic deficiencies. Five of Fowler's patients examined for sludging showed it distinctly. He stated further that all or any one of these factors may be and frequently are the cause of sudden deafness.

In 1950 also, Opheim[7] reported five cases of sudden deafness at the Eleventh Northern Oto-Laryngological Congress. He was much impressed by Mygind's[8] idea, presented at a meeting of the Danish Oto-Laryngological Society, that the loss of hearing might perhaps be brought on by an acute rise of pressure in the labyrinth. Opheim stated that this hypothesis opens a road to a rational intervention against this condition, which otherwise has such an unfavorable prognosis. Fenestration operations were carried out for their decompressive effect on three of his patients. At operation an attempt was made to leave the ossicular chain intact. In all three cases the hearing improved some but not to a serviceable level. On puncturing the labyrinth, the fluid was found to be under pressure in one patient, but not in the other two. Opheim stipulated the following conditions for operation: *1.* The loss of hearing must be complete to the extent that such a condition is recognizable, the function of the other ear being normal. *2.* The patient must be available for treatment at such an early time in the course of disease, that it may reasonably be supposed that the damage to the sensory apparatus has not become irreparable.

Mygind,[9] in discussing Opheim's paper, reported the case of a colleague with sudden deafness. When this patient was admitted to the hospital, his hearing had returned to some extent. Because of this, treatment for Ménière's disease was started, and the hearing became normal. Mygind stated that if the hearing does not return in such cases he would give dextran

(macrodex), a polysaccharide with especially large molecules which cannot pass through the capillary walls.

In 1952, Moulonguet and Bouche[10] reported one case of sudden deafness. The patient, a young doctor, became suddenly deaf in the right ear a few hours after receiving an injection of "orthobiotic" serum. The patient was examined on the fourth day after the injection. The audiogram showed marked loss of hearing in the speech range (65 db). Bone conduction was poorer than air conduction and Fowler's test showed recruitment for the frequencies 512 and 1,024. Moulonguet and Bouche considered that this condition was caused by a vascular disturbance or more precisely an arteriolar spasm. The patient was slowly given an intravenous injection of sodium nicotinate; the usual marked peripheral congestion followed. Three hours later hearing had returned to normal. Administration of sodium nicotinate was continued by the intramuscular route for a week, and the hearing has since remained normal. They commented about as follows: *1.* Because of recruitment, the lesion seemed to be localized to the cochlea. *2.* The spectacular result of a peripheral vasodilating agent confirmed the hypothesis of arteriolar spasm.

MATERIAL STUDIED AND OBJECTIVE OF STUDY.

In the five years from 1949 to 1953, inclusive, 178 patients with sudden deafness of obscure cause were seen at the Mayo Clinic. These cases were studied carefully to determine, if possible, the cause of the deafness. The objective of this study was to determine if possible the underlying cause of the sudden deafness and the result of treatment, if any, with the view of aiding physicians to arrive at the diagnosis.

ETIOLOGY, PATHOGENESIS AND PATHOLOGY.

From the review of the literature, it is evident that generally the etiology of sudden deafness of the obscure type under consideration is unknown, but several possible causative factors were suggested. These briefly are *1.* thrombosis, vascular spasm or hemorrhage; *2.* neuritis of the VIIIth cranial nerve; *3.* systemic disease; *4.* toxic involvement of the labyrinth with infections elsewhere; *5.* emotional episodes; *6.* acute

rise in pressure in the labyrinth; *7.* injection of an "orthobiotic" serum and *8.* Ménière's disease.

Factual information about the pathologic process underlying the loss of function has been impossible to obtain because deafness is seldom or never associated with events that kill the patient.

In 89 of the 178 cases studied deafness seemed to be of vascular origin; in 56 it seemed to be due to Ménière's disease (endolymphatic hydrops); in 17 the cause could not be determined; in 15 it seemed due to miscellaneous causes; and in one case, to hysteria or malingering. The clinical features which led to this decision and any available data concerning diagnosis, differential diagnosis, pathology and pathogenesis will be considered when the types are discussed.

SUDDEN DEAFNESS OF POSSIBLE VASCULAR ORIGIN.

The reasoning behind a diagnosis of vascular accident in a case of deafness of sudden onset is about as follows: The vascular supply of the labyrinth is such that a thrombus, vascular hemorrhage or vasospasm might affect the entire auditory sense organ or only part of it and produce sudden deafness. If the sudden deafness is bilateral, a hydrops type of disorder must be considered because it is unlikely that vascular accidents should occur in both ears at the same time. A history of one attack of sudden deafness with tinnitus in one ear followed in minutes or hours by a dizzy spell, which persists for a few hours to a month but with persistent loss of hearing, is suggestive of a vascular accident. The deafness may often be total and permanent.

Pathologic Hypotheses: Mygind[9] expressed the view that many patients who become deaf suddenly must have hemorrhage into the modiolus. Vestibular symptoms occur only later when bleeding or edema spreads to the nervous system of the labyrinth. The general term "vascular accident" was used by Lindsay and Zuidema.[6] Fowler, Sr.,[1] said that this term was "unscientific;" however, I have used it in this paper, because it implies vascular damage but does not specify the exact type.

Kobrak[3] stated that hemorrhage always should be suspected

whenever deafness develops suddenly, even if there are no signs of hemorrhagic diathesis. If the damage to the inner ear seems to be reversible, transient vasospasm may be an important factor. Occasionally patients have had one severe dizzy spell without any deafness. Subsequent caloric tests often, but not always, reveal a nonfunctioning equilibratory labyrinth on one side. It seems reasonable to assume that in these cases a vascular accident may have involved the vestibular branch of the internal auditory artery only.

A look at the cases shows other features which are compatible with a diagnosis of vascular accident.

TABLE I.

AGE DISTRIBUTION OF PATIENTS WITH DEAFNESS OF VASCULAR ORIGIN AND OF MENIERE'S DISEASE

	Vascular Origin		Ménière's Disease	
	No.	Per Cent	No.	Per Cent
Males	43	48.3	26	46.4
Females	46	51.7	30	53.6
Age, Years				
20-29	9	10.1	2	3.6
30-39	18	20.2	15	26.8
40-49	18	20.2	20	35.7
50-59	21	23.7	12	21.4
60-69	18	20.2	7	12.5
70-79	5	5.6		
TOTAL	89	100.0	56	100.0
Average age, years	49.0		46.3	
Youngest, years	22		20	
Oldest, years	73		66	

Age and Sex: Of the 89 patients who had sudden deafness which seemed to be due to a vascular accident, 43 were males and 46 were females (see Table I). A significant number (27) were from 20 to 39 years old; however, the majority of patients were evenly spread over the older age groups. This age distribution compares fairly well with the age incidence of coronary occlusion. Parker and associates[11] found that 85 per cent of their 3,400 patients with angina pectoris were from 40 to 69 years of age; 64.1 per cent of the 89 patients with sudden deafness of possibly vascular origin in my group were in the same age period. There was a significant discrepancy, however, as only 3.6 per cent of the patients with angina pec-

toris were 20 to 39 years of age, but 30.3 per cent of the 89 patients in this series were in this age group. It is difficult to explain this discrepancy, but one should not forget that branches of the internal auditory artery are end-arteries, so that even minor vascular changes might cause early symptoms.

General Examinations and Pertinent Findings Which Bear on the Cause of the Deafness: The average time from the onset of deafness until the patient was examined at the clinic was four months. Sixty-one (69 per cent) of the 89 patients had general examinations conducted by an internist and 42 patients had a complete neurologic evaluation at the clinic.

Twenty patients had different degrees of hypertension, arteriosclerosis, or both, with hypertensive changes in the ocular fundi. A few were checked for blood sludging as suggested by Fowler, Jr.,[12] but this was abandoned because of variable findings. Rucker[13] expressed the opinion that these might be caused by the heat of the slit lamp. Only two patients had blood dyscrasias: one had polycythemia vera; the other, chronic lymphatic leukemia. Sudden deafness followed a severe transfusion reaction in one case. The patient had been given Rh positive blood, and he was Rh negative.

Hearing: The deafness of all 89 patients seemed to come on without any apparent cause. Ten patients woke up with severe roaring and deafness in one ear. Three patients became deaf while using the phone. Allergic factors or emotional problems did not seem to play a significant part.

All 89 patients except those who were totally deaf had audiograms, speech-reception tests and loudness-balance tests. In 32 patients (36.0 per cent) the deafness was total in one ear. Right and left ears were involved with about equal frequency. Two patients had bilateral involvement; in these two the deafness was total and permanent. The intervals between onset of deafness in the first ear and the second ear were three weeks and one year respectively. The caloric tests revealed nonfunctioning equilibratory labyrinths on both sides. Both patients had compensated well, and were able to move about well, except in darkness.

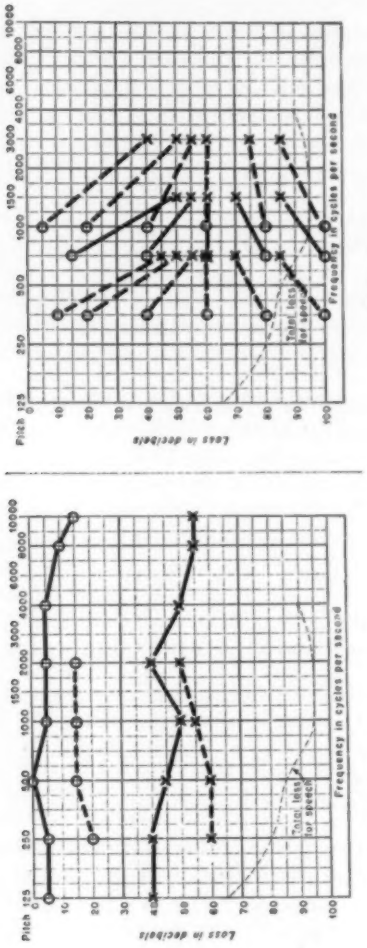

Fig. 1. Case 1. Audiograms and result of loudness-balance test in typical patient with deafness of vascular origin which involved the entire tonal range.

The audiograms of the entire group of 89 patients revealed great variation in the deafness, but it was always of the end-organ type. This diagnosis was confirmed by loudness-balance and speech-reception tests. Thirty-two patients (36.0 per cent) had flat curves with deafness of varying degree. Case 1 is a typical example.

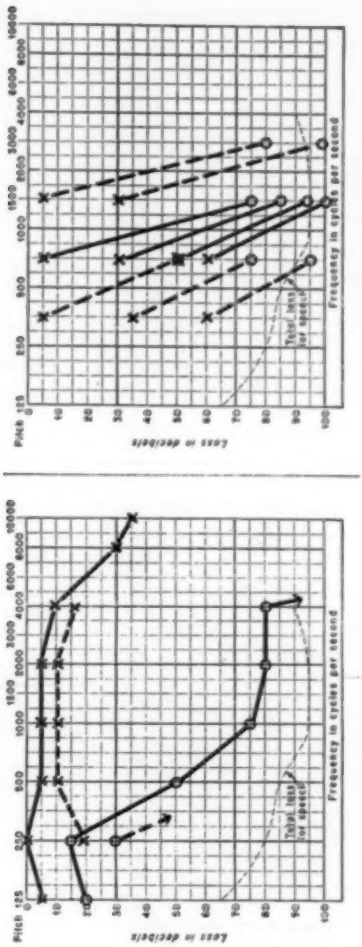

FIG. 2. Audiograms and result of binaural loudness-balance test of patient with deafness of vascular origin involving mainly the hearing of high tones.

Case 1: The audiogram and result of loudness-balance test for a woman 62 years old revealed a moderate end-organ type of deafness with marked recruitment in the left ear over the right (see Fig. 1). Re-examination a year later did not reveal any change in hearing; however, recruitment was much less marked, and there was no evidence of overrecruitment. The patient felt that she could hear better because there was less confusion.

Seventeen patients (19.1 per cent) had mainly loss of hear-

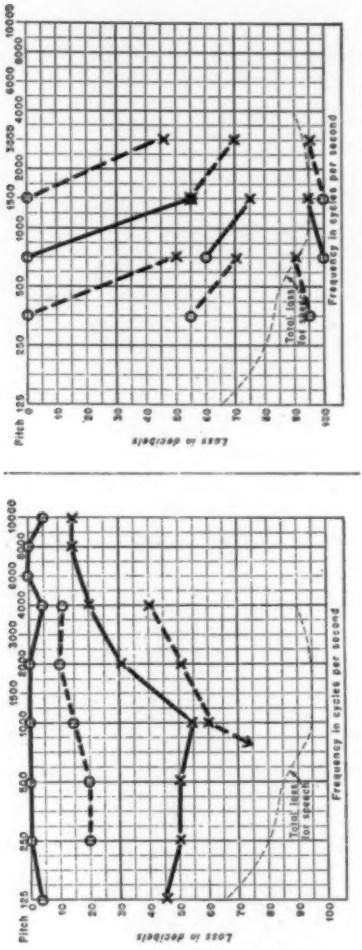

Fig. 3. Audiograms and result of loudness-balance test of a patient with vascular type of deafness involving low tones.

ing for high tones, but the degree of this loss varied. The audiogram and the result of recruitment test of a representative case are shown in Fig. 2. The patient, a woman 59 years old, had suddenly lost her hearing, while playing with her grandchildren. Re-examination two months later revealed no change in hearing. The caloric test revealed normal responses.

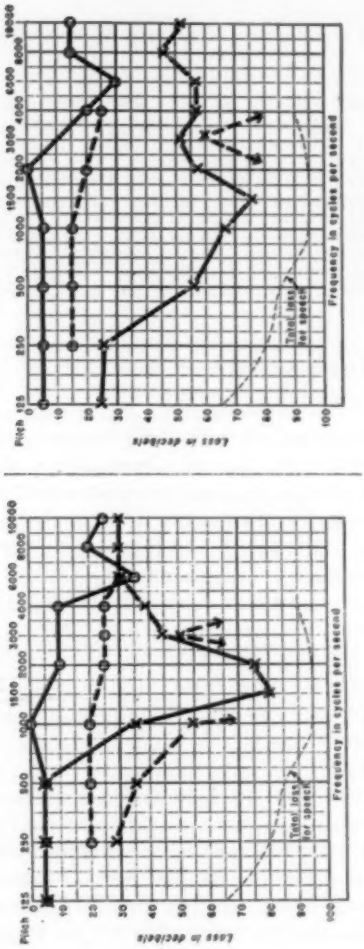

Fig. 4. Case 3. Audiograms of a patient whose sudden deafness started as an island type.

In four patients (4.5 per cent) the low tones were mainly involved. The audiogram and the result of recruitment test of a man, 35 years of age, who had involvement of the low tones, are shown in Fig. 3. Sudden roaring tinnitus had developed in the left ear with deafness but no dizziness five months prior to examination. Hearing has remained unchanged since.

In four patients (4.5 per cent) the deafness was of an island type.

Case 2: One such patient, a woman 48 years old, woke up one morning with severe ringing and deafness in the left ear. She was seen the following morning. Results of the ear, nose and throat examination were negative. The left Eustachian tube was patent. The audiogram (see Fig. 4) revealed a marked dip involving mainly the range of 1,500 to 2,000 frequencies. Treatment was started immediately. She was instructed to take a form of nicotinic acid (roniacol) in tablet form, 100 mg. before breakfast and 100 mg. before supper. Four days later the hearing was definitely worse. Deafness had spread over the neighboring frequencies. When she was examined again one month later, her hearing was essentially normal. The patient was re-examined one and two years later, and the hearing remained normal.

Because of the single attack only, this deafness was felt to be primarily vascular in origin.

Dizziness: In 41 of the 89 patients in this group (46.1 per cent) deafness and tinnitus were the only symptoms. In 48 patients (53.9 per cent), the loss of hearing was associated with varying degrees of dizziness, nausea and vomiting. Usually the vertigo started a few minutes to 12 hours after the onset of the deafness. This could be explained by retrograde thrombosis causing complete or partial blockage of the vestibular branch of the internal auditory artery. More females had dizziness associated with deafness than males. All 48 patients who gave a history of unsteadiness or dizziness at the onset of the deafness underwent caloric tests. About 70 per cent had varying degrees of hypoactivity of the equlibratory labyrinth. Fifteen of the 32 patients with total deafness in one ear and dizziness gave no response to caloric stimulation on the side of the deafness. In the other cases, however, there seemed to be no definite relationship between the degree of deafness and the result of the caloric test. Some of the patients were tested who had no history of dizziness. All had normal responses on both sides, as one would expect. As has been stressed by Lindsay and Zuidema,[6] the sudden onset, the single attack only, and the usually permanent loss of function are sufficient to differentiate the condition from Ménière's disease or hydrops of the labyrinth. In two of our patients the vascular involvement was more extensive. Details of one are given in Case 3.

Case 3: The patient, a man aged 53 years, stated that two months prior to examination sudden total deafness in the left ear, slight dizziness and incomplete facial palsy on the left side developed. Examination revealed total deafness, facial weakness and diminished corneal reflex,

all on the left side. Complete general and neurologic examination otherwise gave negative results.

This patient probably had a vascular accident involving parts of the pons and brain stem, and possibly the posterior inferior cerebellar artery.

Because of all the facts mentioned in this section the pathologic disturbance occurring in the 89 patients classified as having deafness due to vascular accident seems different from that in the next group to be discussed, in which the sudden deafness seemed to be caused by Ménière's disease or hydrops of the labyrinth.

SUDDEN DEAFNESS APPARENTLY DUE TO MENIERE'S DISEASE (ENDOLYMPHATIC HYDROPS).

Etiology, Pathogenesis and Pathology of Sudden Deafness Thought to Be Ménière's Disease: I was unable to find a report of detailed microscopic examination of a temporal bone from a patient with sudden deafness; however, it has been reported that sudden deafness often represents the onset of Meniere's disease. For this reason certain studies on Meniere's disease will be considered here.

The first microscopic observation of the temporal bones of persons known to have had severe attacks of vertigo, deafness and tinnitus during life was reported by Hallpike and Cairns[14] in 1938. They found hydrops of the labyrinth, and it was reasonable to conclude that the effect of this hydrops on the cochlear duct, utricle and saccule was responsible for the symptoms.

Many studies since then have confirmed the histopathologic incidence of an endolymphatic distention of the cochlear duct without inflammatory reaction. As a result of these observations the terms "endolymphatic hydrops," "hydrops of the labyrinth" or "labyrinthine dropsy" are now commonly used to designate this disease.

Although the histopathologic reports are increasing, certain fundamental questions concerning the disease have not been answered. The exact etiology and pathogenesis have not been clearly determined.

Hilger[15] attempted to show that the vital intralabyrinthine structures depend for integrity of function and structure on end-arteries, which do not have collateral circulation. He suggested that a disturbance in the vascular function common to all end circulation can cause vasospastic changes in the labyrinth. These may result in primary neurovascular ischemia and endolymphatic hypertension, which bring about this syndrome. Williams[16] suggested the hypothesis that Ménière's disease is an instance of autonomic imbalance, specifically a cholinergic preponderance or overactivity. He theorized further that Ménière's disease is a form of intrinsic allergy involving the capillary loops of the stria vascularis and that the endolymphatic distention is a localized extracellular edema produced by the spastic-atonic state of the capillaries.

Day[17] stated that in an occasional case a specific protein allergen may prove to be the causative factor and reported observations in several cases in which the symptoms could be abolished by removal of the offending allergens only to recur with their reintroduction into the patient's environment. Day also presented evidence that in labyrinthine hydrops the cochlea is primarily affected and the vestibular disturbance is due to secondary or overflow reaction of the vestibular mechanism.

More recently Lempert and his co-workers[18] have reported on histologic changes in the inner lining of the membranous walls in the endolymphatic external semicircular canals of six patients with Ménière's disease who underwent partial labyrinthectomy for the arrest of persistent dizzy spells. They found that this inner membranous lining of all specimens was studded with vesiculated epithelial excrescences which suggested collections of epithelial fluid. They postulated that an attack of Ménière's disease is due to the rupture of one or more vesicles with release of toxic fluid into the lumen of the endolymphatic membranous labyrinth. They also postulated that Ménière's disease may be primarily a disease of the vestibular labyrinth and that the pathologic changes in the cochlea and the symptoms are the sequelae of vestibular disease. They concluded that the cumulative loss of hearing during the course of the disease is due to progressive damage of the organ of

Corti by the repeated contamination of the endolymph by this vesicular toxic fluid.

Shimamoto and co-workers[19] in 1954 reported some interesting experiments on 160 guinea pigs. They replaced the endolymph with various chemical substances, especially solutions of potassium chloride in various concentrations. They suggested that some disturbance of the secretion or absorption of the potassium of the endolymph may exist in Ménière's disease and if the concentration of potassium could be changed, especially by being increased unequally in both endolymphs, an excitatory syndrome of the vestibular organs would be established just as in Ménière's disease. They considered that it was not so unnatural to speculate that the cochlea might also be affected by an unusually high concentration of potassium in the endolymphatic system.

General Observations on the Group: In the five years under consideration a total of 1,270 patients were seen at the Mayo Clinic and given a diagnosis of true Meniere's disease. The Ménière's disease of 56 patients (4.4 per cent) started with sudden deafness. This figure may in reality be higher since so many of the patients with Ménière's disease are so distressed by the severe tinnitus and dizziness that they do not give the deafness much thought.

Repeated examinations are required in order to permit a correct diagnosis. The history is extremely important. As has been stressed often by Fowler, Jr., and Zeckel,[20,21] emotional factors played an important role in precipitating an attack. This psychosomatic influence seemed, however, to be much more at play *after* the disease had started, and seemed to be a participating factor only in causing repeated attacks. The sudden deafness usually came out of a clear sky and was or was not associated with dizziness. There were remissions and exacerbations of deafness with usual progression of deafness over the years, no matter what type of therapy was used.

Age Distribution and Time of Examination: The age distribution is shown in Table I. Forty-seven (83.9 per cent) of the 56 patients first had their trouble between the ages of 30

and 59 years and only seven patients became deaf after the age of 60 years. The mean age at onset was 46.3 years.

The average time from the onset of deafness until the patient was examined at the clinic was eight months, four months longer than for the first group of patients.

Deafness: As in the "vascular-accident" group all patients were checked with audiograms, speech-reception tests, and loudness-balance tests except where the deafness was total. Here again the deafness varied but was always of the end-organ type. In six patients the deafness was total, or nearly so; in one of these patients the total deafness was bilateral.

Case 4: Another one of these six patients, a man 33 years of age, had suddenly lost his hearing in the left ear at work two weeks prior to examination. Severe dizziness, nausea and vomiting started the following day. Initially, he also had some ringing in the right ear. Dizziness persisted. Ear, nose and throat examination gave negative results; however, the audiogram revealed almost total deafness in the left ear and a moderate amount of end-organ type of deafness in the right. The patient was treated with mono-ethanolamine nicotinate (nicamin), ammonium chloride and low-salt diet for two months, then booster courses were given to ward off symptoms. Audiograms made about one month later revealed no change in hearing on the left side but definite improvement on the right side. The caloric test revealed a nonfunctioning equilibratory labyrinth on the left side and normal responses on the right. On re-examination a year later hearing was unchanged in the left ear, but had improved further in the right.

A surprisingly large number of patients (19) had bilateral deafness. In 13 of these 19 the deafness came on suddenly in both ears and at the same time, and in 12 it was associated with vertigo, nausea and vomiting. In three patients there was an interval of from six hours to a month between the sudden onset of deafness in the first and the second ear. In one patient the interval was nine years. In two patients the onset of deafness was gradual in one ear and sudden in the other. In most of these patients hearing fluctuated after the initial attack.

Generally speaking, the more severe the deafness was at onset, the more likely there was to be bilateral involvement and the less chance for benefit from treatment. Involvement of the entire tone range also seemed to lessen the chance of benefit from treatment. Seventeen patients (39.0 per cent) had involvement of the entire range of tones. Case 5 is a typical example.

Case 5: The patient, a man 50 years of age, became deaf suddenly five months before he was examined at this clinic, and in the intervening months before admission had had several severe dizzy spells. At the clinic the regular Mèniere's disease regimen was instituted with salt-poor diet and doses of nicotinate (nicamin) and ammonium chloride. When the patient was re-examined three months later he stated that he felt better, had had no further dizzy spells and felt his hearing had improved. The audiogram and loudness-balance test revealed, however, that the hearing was unchanged. The loudness-balance test still showed recruitment but it was much less marked. This finding means less distortion of speech.

None of the 10 patients (61 per cent) with involvement of the entire tonal range who were re-examined had any real improvement of hearing, as indicated by audiograms and speech-reception tests; however, slight variations in hearing from day to day were noted.

The patients who had sudden deafness involving only part of the tonal scale would have fluctuation in their ability to hear, and 14 (about 40 per cent) ended with normal or nearly normal hearing with or without treatment. Of these, seven patients had mainly a loss of high tones; five patients had mainly loss of low tones and only two had deafness which was confined to the speech range. One patient (Case 6) is of special interest because she had lost ability to hear high and low tones in the same ear, but these changes came on at different times.

Case 6: The patient, a woman 37 years old, lost her hearing in the right ear suddenly while she had both hands in cold water; a roaring tinnitus and "numb feeling" occurred in the right side of the occiput. Two days later a severe dizzy spell associated with nausea and vomiting developed. The audiogram taken five days later showed a moderate loss of hearing for high tones of end-organ type with marked recruitment. Caloric tests revealed normal responses on both sides. She was treated with monoethanolamine nicotinate (nicamin), ammonium chloride and salt-poor diet. Within a week the hearing returned to normal. About a week later, she again lost the hearing in the same ear (right) suddenly. This episode was also accompanied by dizziness and nausea. This time the low tones were also involved.

Audiograms and a loudness-balance test were made six days later and an audiogram two months later. By that time her hearing was essentially normal.

Dizziness: The dizziness varied from a transient unsteady feeling to a violent vertiginous attack associated with nausea and vomiting. Usually the more severe the associated dizzy spell, the more it would show up in the result of the caloric test.

Only 16 patients (28.6 per cent) did not give a history of any dizziness. The remaining 40 (71.4 per cent) gave a history of dizziness of varying intensity. In most of them the dizziness came on either at the same time as the deafness or a few hours to two days later. In five patients the vertigo was the initial symptom.

In the six patients who had total or nearly total deafness, there generally was marked depression of caloric function. In 36 of the 40 patients who had dizziness associated with deafness, the equilibratory labyrinth was hypoactive or nonfunctioning. In four of the 40 patients responses were either within normal limits or hyperactive.

The carefully elicited general history did not give any definite clues as to what might have precipitated the hydrops. There did not seem to be any definite allergic relationship and only nine (16.1 per cent) had hypertension, arteriosclerosis or both with sclerosis of the retinal arteries of varying degree. My colleagues and I were unable to obtain a definite history of emotional upsets at the onset; however, they seemed to be important in precipitating subsequent dizzy spells with or without further deterioration of the hearing.

DEAFNESS OF UNDETERMINED CAUSE: HYDROPS OR VASCULAR ACCIDENT.

In 17 cases the diagnosis could not be made definitely. Most of these patients were seen only once or were seen too recently to permit evaluation of the possible cause.

SUDDEN DEAFNESS OF MISCELLANEOUS ORIGIN.

Deafness of sudden onset, sometimes associated with vertigo, is known clinically to occur on a toxic basis as a complication of some infectious diseases, which are mainly virus in type. Lindsay and Zuidema[6] stated that there is no constant pattern with regard to the proportion of hearing loss that is due to vestibular dysfunction as a result of a toxic process.

In most of the 15 patients in this group the deafness was considered as toxic in origin. The youngest patient was six, the oldest 74 years of age. About half of the patients were

between 40 and 49 years of age. All 15 patients in this group had complete general and neurologic examinations, audiograms, loudness-balance tests, speech-reception tests and caloric tests, if deafness was associated with dizziness.

Deafness: As in the first two groups the deafness varied from total bilateral to moderate unilateral deafness. The deafness was of an end-organ type with evidence of moderate to marked recruitment in all except two patients. One of these two who had no recruitment had multiple sclerosis, and the other had a tumor of the cerebellopontine angle.

TABLE II.

SUDDEN DEAFNESS OF MISCELLANEOUS ORIGIN

Intimate Relationship or Cause of Deafness	Patients
After operation and general anesthesia	2
Heavy dosage of aspirin for arthritis	2
Syphilis	2
Peptic ulcer, abscess and antibiotics	1
Severe acute upper respiratory infections	2
Large dosage of alcohol and bromoquinine	1
Herpes zoster	1
Booster shot for whooping cough (case 7)	1
Multiple sclerosis (case 8)	1
Cogan's syndrome (case 9)	1
Cerebellopontine-angle tumor (case 10)	1
TOTAL	15

As might be suspected, in eight of 15 patients (53.3 per cent), the sudden deafness was bilateral, and in three the deafness was total.

The deafness of only five patients was associated with dizziness. All five had bilateral loss of hearing. This observation tends to confirm the statement that the equilibratory apparatus is much more resistant to toxic influences than the cochlea. After the initial loss, the hearing remained down in 13 patients. In the other two, congenital syphilis was thought to be the cause of the toxic effect. The hearing of one of these two patients fluctuated rather markedly after antisyphilitic treatment, but two months after the treatment was started the hearing remained down. In the other patient, in spite of initial fluctuation in hearing, the hearing in the left ear disappeared entirely.

The cause of the toxic effect, elicited from the history, was varied and difficult to nail down exactly. The diseases or conditions which were active or present at the onset of the sudden deafness in these 15 cases are presented in Table II.

The last four cases (Cases 7 to 10) referred to in Table II are unusual and deserve further comment.

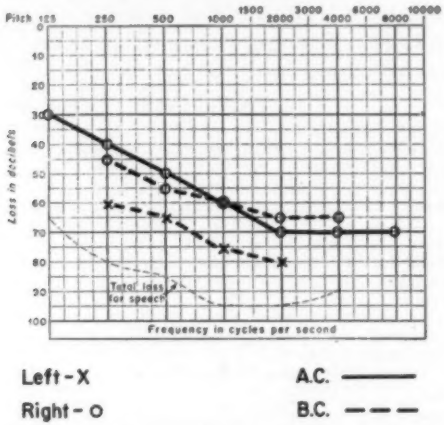

Fig. 5. Case 7. Audiogram of patient with sudden deafness from whooping cough-booster injection.

Booster Injections for Whooping Cough.

Case 7: The patient, a girl aged six years, was first seen Aug. 4, 1951. Her mother gave the following history: Her sister had had whooping cough and the local physician advised booster shots for the patient. On the way home from the doctor's office, a 25-mile drive, the patient said her ears felt "funny." As soon as they got home the girl said she could not hear a thing. The patient vomited at first but did not stagger; her personality changed and temper tantrums and overactivity developed. The patient was seen two and half months after the onset of deafness. Results of the ear, nose and throat examination were essentially negative. The first attempt to check the hearing with audiograms did not seem accurate. The testing was done again a few days later without the parents being present (see Fig. 5). The child then was much more co-operative.

A hysterical reaction had to be ruled out. The patient could not tolerate electrodes for galvanic skin tests. Nothing definite was found on complete neuropsychiatric checkup except oversolicitous parents. Since the onset of the deafness there had been no definite fluctuation in hearing, and the child had already become an efficient lip reader. Active

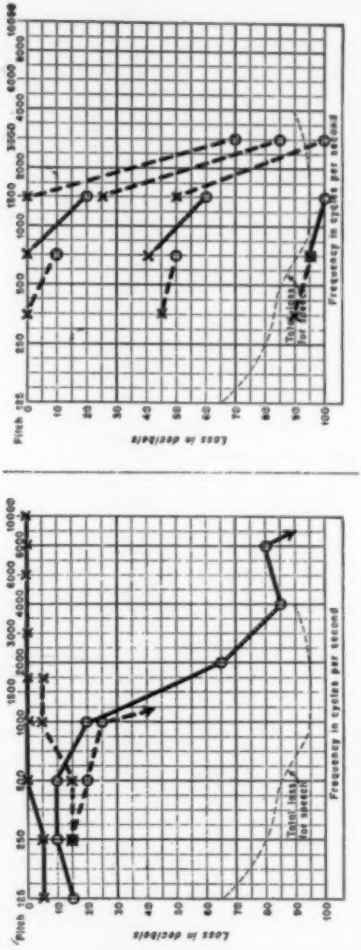

Fig. 6. Case 8. Audiograms and recruitment test of patient with multiple sclerosis.

training at home was suggested, and the parents were advised to contact the John Tracy Institute for home training programs. The caloric test was not done; however, the patient had not experienced any dizziness or had any trouble in walking and playing. The final opinion was that the sudden deafness was probably caused by an anaphylactic phenomenon either involving the nerve or cochlea. Because of poor speech reception on the right side, indicating distortion, the cochlea itself probably was affected.

I was unable to find a similar case in the literature. Sudden deafness following tetanus antitoxin, however, has been reported. An excellent description and bibliography were given by Berger and Sachs[22] in 1953. Their case was the third reported in the literature. In the three cases reported in the literature the deafness came on about a week after the injection during an eruption of serum sickness (generalized urticaria).

Sudden edema of the labyrinth is probably responsible for the deafness due to an anaphylactic reaction; however, the knowledge about deafness caused by allergy, serum sickness and anaphylaxis is not complete enough to permit a statement of the entire true picture of the pathology.

Multiple Sclerosis.

Case 8: Two years before the patient, a woman aged 27 years, was first seen, a roaring sound had developed suddenly in the right ear, which had felt completely "blocked up." The roaring lasted two weeks and then subsided, but deafness persisted. No dizziness was noted. The doctor in the home locality gave her histamine, but it did not help. The hearing never seemed to fluctuate. For about two months she had spells of slurring speech, transient numbness of the left side of the body and some "dragging" of the left leg. Examination of the ears, nose and throat gave essentially negative results. The Eustachian tubes appeared to be patent. The audiogram revealed marked deafness for high tones on the right and binaural loudness-balance test showed significantly no recruitment (see Fig. 6).

The caloric responses were normal on both sides. The significant ocular findings were: *1.* spontaneous vertical nystagmus on looking up and *2.* sheathing of the retinal veins, which according to Rucker[23] is found in about 10 per cent of patients with multiple sclerosis. General examination was noncontributory; however, the neurologic examination revealed some numbness and weakness of the left leg. A diagnosis was made of multiple sclerosis. Because the deafness was of a nerve type with no evidence of recruitment, it seemed to be caused by the same disease process. It seemed probable that the disease attacked the auditory nerve itself, corresponding to optic neuritis, which is commonly found in multiple sclerosis.

In 1888 Hess[24] reported auditory findings in a case of multiple sclerosis which he had observed for more than eight years. Sudden bilateral deafness became permanent in the right ear, while the hearing in the left ear returned gradually after 24 hours. Sudden loss of hearing associated with multiple sclerosis is rare. Apparently none of the 92 patients studied by Von Leden and Horton[25] experienced sudden deafness. These investigators found, however, that defects in the

auditory field, comparable to defects in the perimetric field, occur fairly frequently.

Bentzen and associates[26] stated that in most cases of multiple sclerosis in which impairment of hearing has occurred, it has been transitory, being mentioned usually as a link in acute pontobulbar outbursts. A continuous analysis of 64 patients revealed a considerably higher auditory activity of the disease

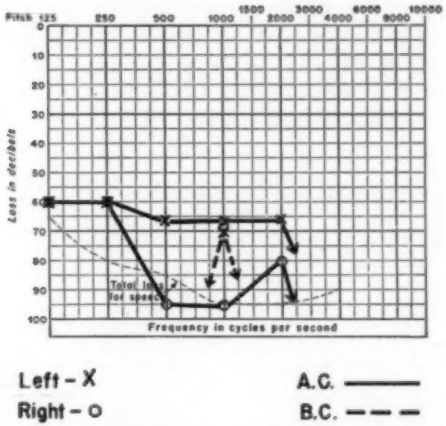

Fig. 7. Case 9. Audiogram of patient with Cogan's syndrome.

than is usually reported. A fifth of the patients had impairment of tone perception. The acoustic disturbances (including tinnitus) caused marked subjective discomfort in only a few.

Phillips[27] stated that symptoms referable to the ears were outstanding events at the onset of multiple sclerosis in two per cent of cases and the most frequent such symptom is vertigo. Phillips expressed the opinion that the deafness is caused by actual involvement of either the auditory nerve or the cortex of the brain in the auditory radiation. Krohn,[28] of the University of Oslo, Norway, told me that he had never seen a case in which sudden deafness was an outstanding symptom.

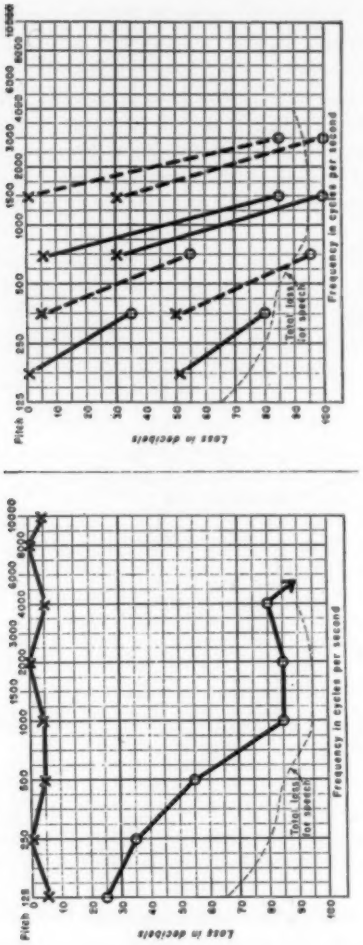

Fig. 8. Case 10. Audiograms and result of loudness-balance test of patient whose deafness was due to a tumor of the cerebellopontine angle.

Cogan's Syndrome.

Case 9: The patient was a 27-year-old man who, five months before the examination, was dizzy one morning on arising. He also felt nauseated but did not vomit. Three days later sudden bilateral tinnitus and deafness developed. He said he could not hear; however, hearing returned gradually within the next few days, but deafness returned suddenly three months later. Dizziness disappeared, but now the deafness re-

mained. At about the same time the eyes became reddened, and the redness persisted.

Except for profound deafness the ear, nose and throat examination gave negative results (see Fig. 7). Caloric tests revealed nonfunctioning equilibratory labyrinths on both sides. Examination of the eyes revealed corneal opacities of a vascular type, presumably in the interstitial layers of the cornea. Results of the general examination and of flocculation tests for syphilis were negative.

This was evidently the syndrome of nonsyphilitic interstitial keratitis with vestibular auditory symptoms first described by Cogan[29] in 1945. The patient was treated with aureomycin and histamine for a month. Reexamination and audiogram then showed no change.

Cogan's syndrome usually occurs in young adults and is often of sudden onset, either with ocular or aural symptoms. According to Oliner and associates,[30] symptoms from eyes and ears may occur within a few hours of each other, or at any interval up to five months. In the majority of cases the interval is between one and two weeks. Lindsay and Zuidema[6] reported three cases of this type with sudden loss of hearing. In all their cases, as in mine, the deafness was bilateral. The etiology is not known; however, the inflammatory nature of the disease seems definite.

Cerebellopontine-Angle Tumor.

Case 10: The patient, a doctor's wife, 46 years of age, first was seen in 1947 for a pelvic operation. She returned in March, 1953, for a routine examination and for a check of hearing because of deafness on the right, which had come on suddenly two years before while she was walking over a frozen lake. Roaring tinnitus was associated with the deafness at first but subsided in a couple of weeks. The deafness, however, became gradually more severe. She would not have consulted the clinic for this if she had not accompanied her husband.

Results of ear, nose and throat examination were essentially negative. The audiogram (see Fig. 8) revealed a marked nerve-type deafness on the right, and the binaural loudness-balance test showed conspicuous absence of recruitment. The caloric test revealed a nonfunctioning equilibratory labyrinth on the right. The corneal reflexes were normal on both sides; however, a fine rotatory nystagmus was present on lateral gaze to the left. The quick component appeared to be clockwise.

Except for these findings the neurologic examination was entirely negative, and there was no evidence of choked disks. Roentgenograms of the head, including Stenver's views, were negative. The only other significant finding was increased protein (190 mg. per 100 cc.) in the spinal fluid. General examination showed mild hypertension.

On the strength of the rather meager findings the right cerebellopontine angle was explored and a neurofibroma 2.5 cm. in diameter was removed in pieces. Incomplete facial palsy developed on the right side, but otherwise the patient's postoperative course was uneventful.

Sudden deafness associated with cerebellopontine-angle tumors is rare. Cushing[31] reported on the sudden onset of deaf-

ness of the left ear associated with violent vertigo. Most patients with tumors of the cerebellopontine angle do not have real vertigo. Both Cushing's and my cases probably represent a vascular accident involving the blood supply to the nerve which was brought on by encroachment of the tumor.

PSYCHOGENIC DEAFNESS (EMOTIONAL DEAFNESS).

I tried to exclude the possible influence of psychogenic deafness on results by keeping in mind the differential diagnosis

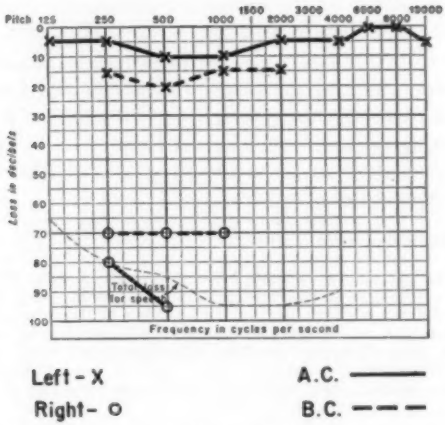

Fig. 9A. Case 11. Audiogram of patient with possible emotional deafness.

between this type and other types of perceptive deafness. Psychogenic deafness is caused by a combination of psychic and organic factors; the latter may be any kind of hearing loss. There are certain clues for detection and Doerfler[32] listed the following: *1.* sudden onset of bilateral severe hearing loss, perceptive in nature; *2.* saucer-shaped curve in audiogram (50 to 90 decibels) in 80 per cent of his cases; *3.* unexplainable discrepancy between audiometric results and tests for hearing of speech, especially when the hearing for speech is better than that for pure tones; *4.* surprising response to therapy out of proportion to what may reasonably be expected, and *5.* results

of special tests such as Doerfler-Stewart test or galvanic skin response.

Blegvad[23] added other symptoms suggestive of psychogenic deafness such as agitation and anxiety during interviews and variation in the tests on different days. Blegvad also stated that these patients usually have flat curves in the audiograms.

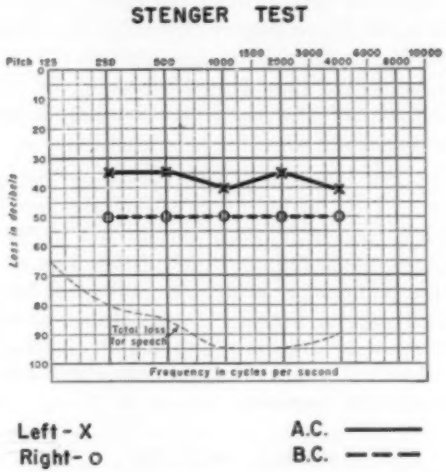

Fig. 9B. Case. 11. The Stenger test. Top curve represents threshold in left ear with each tone given simultaneously at 50 decibels in right. This appears to be a definitely positive finding on Stenger test.

Only one patient in my series had deafness that was due to either hysteria or malingering.

Case 11: A young married woman, aged 23 years, was in an auto accident. The next day she suddenly lost hearing in the right ear. She had no history of aural hemorrhage or dizziness. She was examined two months later. The ear, nose and throat examination gave essentially negative results. The audiogram revealed practically total loss of hearing for pure tones on the right (see Fig. 9A). The result of the Stenger test (see Fig. 9B), however, was considered positive.

The patient had an insurance claim, and the case had not been settled as yet. We do not know what ultimately happened to the patient's hearing.

One can only conclude from this study that, in clinical practice, psychogenic or emotional deafness of sudden onset must be rare indeed.

DIFFERENTIAL DIAGNOSIS IN GENERAL.

As mentioned earlier in this paper, sudden deafness of more or less obvious cause is not included; however, the possibility of the deafness[2] being caused by a catarrhal otitis media should be kept in mind. Often in these patients the tympanic membrane may look entirely normal or may be thickened so that no fluid is seen. The deafness, however, is always of the conduction or combined type. Often the patients give a history of having an acute upper respiratory infection at the onset of the deafness. It was surprising and disconcerting to see how many of the patients with sudden deafness of the obscure type in my series had had prolonged treatment with inflations of the Eustachian tube.

Whether sudden deafness may be caused by hydrops or vascular accident or possibly both may be impossible to determine, especially if deafness is of recent onset. One of the outstanding diagnostic points is found in the type of onset and whether the deafness is bilateral or unilateral. Bilateral onset is more indicative of Ménière's disease; the unilateral, of vascular accident. Only with careful examinations which include consultations with medical and neurologic specialists can one hope to arrive at a specific diagnosis.

GENERAL COMMENT.

Sudden deafness may be the result of any one of several conditions affecting the inner ear for which the etiology is not clearly proved. For this reason it has been a favorite area for speculation and development of theories which have been used as a basis for therapeutic procedures.

Patients who are seen immediately after the onset of deafness should be treated vigorously for Ménière's disease in the hope that irreversible changes have not already taken place in the cochlea. If hydrops is suspected to be the cause, repeated "booster treatments" seem to be of benefit.

Treatment of patients who have been deaf for two months or more and who have had no fluctuation in hearing seems useless. The results of treatment in the present series as far as hearing is concerned have been disappointing, and at least one

of the reasons for this might be that the patients presented themselves late for treatment. In such a fine mechanism as that of the end-organ of hearing, irreversible changes may take place quickly. Rasmussen[5] considered the prognosis poor for the acoustic function, but favorable for the vestibular function. Hallberg and Horton[34] reported four cases of deafness in 1947 in which treatment with histamine was employed with striking improvement of hearing in one. They called the deafness "sudden nerve deafness," which is obviously a misnomer; it should have been named "sudden deafness of end-organ type."

Audiograms, loudness-balance and speech-reception tests should always be made on a patient who gives a history of sudden deafness. Of the diagnostic tests, those for hearing give the most differential information. Of greatest value are the test for diplacusis, the loudness-balance test for recruitment, and the discrimination test for phonetically balanced words.

SUMMARY.

A study was made of 178 cases of sudden deafness of obscure cause encountered in a five-year period, 1949 to 1953, inclusive. In 89 cases the deafness seemed to be due to a vascular accident. In 56 it seemed to be due to Ménière's disease. In 17 the cause could not be determined. In 15 it was due to miscellaneous causes usually of infectious origin. In one case the deafness was thought to be due to hysteria or malingering. The literature, pathology, etiology, diagnostic factors and other pertinent data were considered in some detail. Because of the absence of specific information as to the underlying pathology, the diagnosis as to etiology has to be somewhat tentative; however, in most of the cases studied, repeated and extensive examinations permitted a statement as to etiology which is considered fairly authoritative.

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NEW LOS ANGELES BRANCH FOR V. MUELLER & COMPANY.

A new retail branch store will be opened by V. Mueller & Company, Chicago, at 747 S. LaBrea, Los Angeles, Calif., the first of November. Designed to augment and improve services to West Coast surgeons and hospitals, the new branch will maintain a most comprehensive line of standard and special surgical instruments, in addition to supplies and equipment, plus direct teletype connection with the Chicago warehouse. The new operation under the direction of Mr. Hal Markham, long-time representative of V. Mueller & Company in the Los Angeles area, becomes the fifth Mueller branch strategically located about the West and Midwest. Others are in Chicago, Dallas, Houston and Rochester, Minnesota.

RECONSTRUCTION OF THE HYPOPHARYNX AND CERVICAL ESOPHAGUS AFTER RADICAL EXCISIONAL SURGERY.*

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Syracuse, N. Y.

INTRODUCTION.

Otolaryngologists are performing more radical head and neck operations each year. What is radical head and neck surgery? Generally speaking, it is the treatment of malignancies in this area with the knowledge and skill necessary to control effectively a primary lesion, a cervical metastasis, or both. *In addition,* it is the knowledge and skill necessary to restore effectively that which has been removed, so that the final result is cosmetically and functionally satisfactory for the patient. It is a combination of otolaryngeal, maxillofacial, and plastic surgery.

Recently, emphasis has been placed on excisional surgery as the primary treatment of head and neck cancers.[1,2,3,4,5] This has been greatly helped by improved anesthesia, unlimited blood replacement, and the use of antibiotics and chemotherapy. As radical surgical procedures increase, so do major problems of reconstruction. It is on these growing problems of reconstruction that increased attention should be placed, for this particular phase of head and neck surgery could be improved to rehabilitate the patient more completely. This is true in smaller communities especially, where resistance to radical surgery is based upon knowledge of a case in which reconstruction was not attempted or in which reconstruction failed.

This paper will review reconstruction techniques following radical surgery of the hypopharynx, larynx, and cervical

* Submitted as Candidate's Thesis to American Laryngological, Rhinological and Otological Society, Inc., 1956.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication Jan. 20, 1956.

esophagus. Two methods which have not been described previously and have been used successfully by us will be discussed, as well as other related problems in reconstruction.

REVIEW OF HISTORY.

The pioneer surgery for the removal of cancer of the hypopharynx and the cervical esophagus began in the Nineteenth Century when Mikulicz[6] and Czerny[7] successfully excised a carcinoma from this region. Mikulicz actually made an attempt to reconstruct the defect left from his excisional surgery. This patient died 16 months later from recurrent carcinoma. Mortality on operating was 70 per cent at the turn of the Century. Seventy-five per cent of these cases surviving surgery died of recurrent disease.[8]

In 1908 Von Hacker reviewed the literature (25 cases) and reported that his mortality rate was 48 per cent;[9] he also introduced a two stage method of closure whereby a skin lined esophagus was fashioned from two lateral rectangular flaps.

Lesions in the hypopharynx and the cervical esophagus, as stated by Lane in 1911, were one of the more challenging and difficult problems in surgery; for often a patient appeared to suffer more discomfort after surgery than before.[10] Lane attempted to repair his excisional defects by utilizing a horizontal skin flap with the bases in the left neck.

In 1913 surgeons were performing "neck gland resections" to remove large epilaryngeal lesions.[11-12] It took courage in those days to do this surgery, even though they were actually suprahyoid node dissections. If the lesion were on the anterior portion of the hypopharynx or cervical esophagus, the vertical limb of the initial incision was made as far posteriorly or laterally on the neck as possible and still obtain an exposure to the lateral pharynx; then the anterior flaps were used to cover the defect on the anterior wall of the esophagus. Conversely, if the lesion were located on the posterior pharyngeal wall the initial vertical limb of the incision was made as near the midline as possible so the skin flap could be used to cover the posterior defect in the first reconstructive stage of the lateral transthyroid pharyngotomy.[13,14] It is interesting to note that during this era emphasis was placed more on the recon-

struction phase than on the excisional. For this reason initial excisional surgery often was inadequate due to the surgeon's desire to preserve tissue for reconstruction.

In 1925 Eggers reported his first case where surgical excision was used successfully.[15] No attempt, however, was made at reconstruction. It was during this period that surgeons became radical enough to expect a three year cancer cure; but still a good method for reconstructing the esophagus was lacking. Reconstruction often was prevented by complications, such as infection, shock, or lack of knowledge.

In 1926 Mackenty, a definite pioneer in surgery of the larynx, made the following statement: "I have put behind me an ambition of securing primary union." This was an expression of the times.[16]

Watson reported his first cervical esophageal case with reconstruction in 1940.[17] This patient was reported alive and well eleven years later. The esophagus was mobilized, and the lesion was exteriorized; a skin flap was then placed posteriorly to the esophagus and, during a second operation, the carcinoma was excised. A new cervical esophagus was fashioned later by further plastic surgical procedures. Watson admitted, in the critique of his method, that although successful in this instance, this method did fail in several subsequent cases. This type of surgery was not radical enough to insure against metastases to the cervical and upper mediastinal lymph nodes, and in the later cases there were recurrences at the upper or lower margins of the esophageal section and invasion of the disease into the larynx.

CURRENT TRENDS.

From 1940 to 1950 Watson, Wookey, Orton, Martin and colleagues contributed heavily to the extension of excisional surgery so that more cases were alive to be considered for reconstruction.[5,16,17,18,19] The advancement of this type of surgery was due to the courage of these men and their ability to reduce postoperative complications.

There is more agreement among the surgeons doing head and neck work today than ever before. Many believe that a

minimum of surgery can be offered sometimes if the diagnosis is early; however, in most cases, where the diagnosis is late and the disease is advanced a maximum of surgery is absolutely necessary.

The patient often is left with a large pharyngeal skin fistula with saliva flowing freely onto the skin of the neck. Until this defect is repaired he is unable to swallow, must feed with a nasal gastric tube, and speech is unintelligible. If a laryngectomy is combined with excisional surgery the patient cannot attempt an esophageal voice. During this period many pa-

TABLE I.

SUMMARY OF RADICAL EXCISIONAL OPERATIONS MENTIONED IN LITERATURE.*

1877-1955.

	Cases**
A. Partial pharyngectomy and/or cervical esophagectomy conserving the larynx.	1
B. Partial pharyngectomy and/or cervical esophagectomy with total laryngectomy.	1
C. Partial pharyngectomy and/or cervical esophagectomy, total laryngectomy with thyroid lobectomy combined with radical neck dissection.	4
D. Partial pharyngectomy and/or cervical esophagectomy, total laryngectomy, total thyroidectomy combined with bilateral neck dissection.	1
E. Total pharyngectomy and/or total cervical esophagectomy, total laryngectomy and total thyroidectomy.	4
F. Total pharyngectomy and/or total cervical esophagectomy, total laryngectomy, total thyroidectomy combined with radical neck dissection.	4
G. Total pharyngectomy and/or total cervical esophagectomy, total laryngectomy, total thyroidectomy combined with bilateral neck dissection (either one or two stages).	9
TOTAL	24

* References: 1-19.
** Our group.

tients become critically depressed. Although his malignancy often is controlled or "cured," it means little to him while his swallowing mechanism is out of order.

Many of these defects are repaired by a simple layer closure. When bilateral neck dissections have been performed, and there is a good deal of postoperative scarring of the neck, and in some cases with scarring effects of heavy radiation therapy, the problems appear extreme. It is in these cases that the abil-

ity, training and experience of the surgeon make the difference. It is here where the individual problem presented often is solved by the ingenuity of the operating surgeon.

Table I represents various procedures described in the literature which are between the maximum (for advanced cases) and the minimum (for the very early cases). Table II represents the methods of reconstruction which have been described in the current literature together with two new methods which

TABLE II.

SUMMARY OF METHODS OF RECONSTRUCTION MENTIONED IN LITERATURE.*

1908-1955.

		Cases**
A.	Anastomosis with neck flexed or transverse closure	7
B.	Skin tube and double sliding flaps.	3
C.	Migration of tube pedicles.	5
D.	Wire mesh-skin graft.	2
E.	Migration of flat pedicle.	1
F.	Use of tongue as pedicle.	1
G.	Died before reconstruction.	5
	TOTAL	24

* References: 9, 13-15, 17-19, 22-30.
** Our group.

I used successfully in two instances. Each will be described in detail. The advantages and disadvantages of each will be discussed together with my observations in using these procedures.

SURGICAL METHODS.

End-to-End Anastomosis is the least complicated method, technically, and the one most frequently used to effect reconstruction. It usually is successful if used when indicated, as in cases where the primary lesion is in the hypopharynx or extrinsic larynx and has spread by direct extension to the uppermost part of the cervical esophagus. The spread to the cervical esophagus in these instances is usually an early, well defined, direct extension of the original lesion and appears resectable by including a segment of the cervical esophagus. A radical neck dissection may or may not be combined with this procedure depending upon the circumstances.

The repair used in these cases is the same as used by general surgeons in the repair of intestinal defects when a segment of small or large bowel has been resected. Its advantage lies in the principle of the transverse closure which prevents stenosis and complicating strictures after the healing is complete. For many years laryngologists closed these hypopharyngeal defects in a vertical line. This lessened the potential diameter of the repaired esophagus, since the greatest circumference at the point of reconstruction could never exceed the width of the mucous membrane left on the posterior esophageal wall. This was recognized by Jackson, Clerf,[20] and Cunning[21] who utilized a "T" closure. The "T" closure increased the diameter at the point of reconstruction and later evolved into a horizontal or transverse closure which gives the greatest diameter. Laryngologists today are very successful in effecting a closure even when as little as ½ cm. of mucosal strip remains. Usually these heal without the complication of fistula formation.

The utilization of the transverse closure enables surgeons to increase the potential diameter at the time of closure so it is rarely ever smaller than the original esophageal diameter. It is closed in layers with a 3-0 or 4-0 atraumatic chromic intestinal suture. A continuous suture is placed inverting each stitch so that the mucosa is always turned towards the lumen and approximates itself. This makes a tight seal. The first suture line then is reinforced by a second layer of interrupted sutures covering the entire length of this first suture layer.

If it is not possible to save a mucosal strip[22] then the direct end to end anastomosis is necessary. This is possible in selected cases where the neck is thin and the surgeon is able to mobilize the distal segment and pull it superiorly so that a direct transverse anastomosis of the distal proximal ends of the esophagus is obtained. A flexing of the neck greatly aids in closure. With the help of the orthopedic department we have made a light head case to hold marked flexion for seven days and found that primary healing was greatly facilitated. If this anastomosis fails, as it could if the resected segment of the cervical esophagus is more than 2 cm., it still will have held for a minimum of five to six days. By this time the radical neck flaps are well sealed and reconstruction is far less

difficult than if the alternate method of closing with pharyngostoma and esophagostoma were used. A disadvantage of this alternate method is that salivary secretions will flow over the fresh neck wound and primary healing of the skin flaps often is delayed. Some cases will close spontaneously if the complicating salivary fistula is not too large, and the distal drainage is not impeded by stenosis at the point of anastomosis.

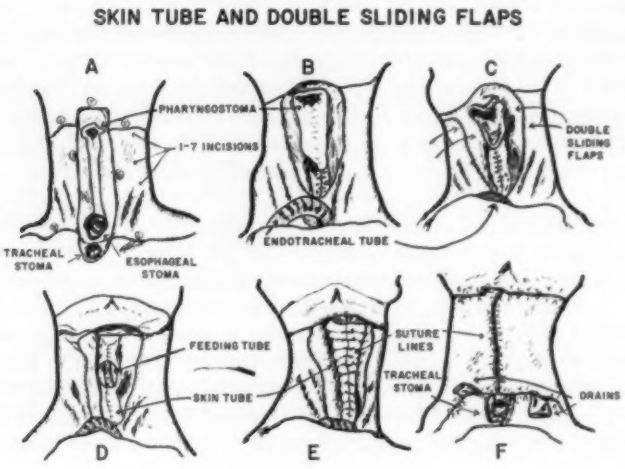

Fig. 2

The Skin Tube With Double Sliding Flaps, as described by Watson,[23] is today one of the most used reconstruction procedures of head and neck surgeons. Its application is broad, and the final result usually is excellent. It is used when excisional surgery leaves a pharyngostoma and an esophagostoma. The skin between the pharyngostoma and the esophagostoma is healthy enough to withstand mobilization and multiple incisions. (See incisions 1 to 7 as diagrammed in Fig. 2A. The central island of tissue left connecting the pharyngostoma with the esophagostoma is rolled onto itself (see Fig. 2B). Then the two lateral rectangular flaps are well undermined

(see Fig. 2C); a feeding tube is put in place, and the first row of sutures is completed (see Fig. 2D). Fig. 2E shows the reinforcing line of sutures over the initial suture line. The two lateral rectangular flaps are mobilized in such a way that it is possible to bring them to the midline and close them under tension. Drains are placed (see Fig. 2F). It is important that one of the rectangular flaps be made slightly longer than the other in the final reconstruction so that the outside suture line is located to one side of the inner suture line and not in exact contact. This lessens the possibility of fistula formation.

The advantages of this method of reconstruction are obvious. In a one-stage technique it usually is possible to undertake and complete reconstruction three to four months after the original excisional surgery. Often it could be done before this time, but it seems wise to wait to be certain that residual tumor is not present. The waiting period is advantageous, too, since the skin quality improves during this interval and increases the likelihood of successful reconstruction.

This method can not be used when secondary healing from a postoperative wound infection causes marked scarring in the neck. The tissues in the neck then may be of such a quality as not to withstand the amount of plastic work necessary in this one one-stage procedure. This particularly is true if the patient has undergone a laryngectomy followed by a bilateral radical neck done in two stages. This type of surgery leaves the neck with little reserve tissue. Heavy radiation prior to excisional surgery is also a contraindication to the use of this method. When the surgeon is faced with either of these situations it is best to consider the migration of healthy tissue to the neck from a distant donor site such as the chest or abdomen. This method we will discuss next.

The Use of a Single or a Bilateral Double Tube Pedicle Graft[24] to bring good tissue from an hairless portion of the anterior chest to the neck has played an important part in the reconstruction of many of these cervical, esophageal, and hypopharyngeal defects during the past several years (see Figs. II, III. Many of these defects could not be closed before

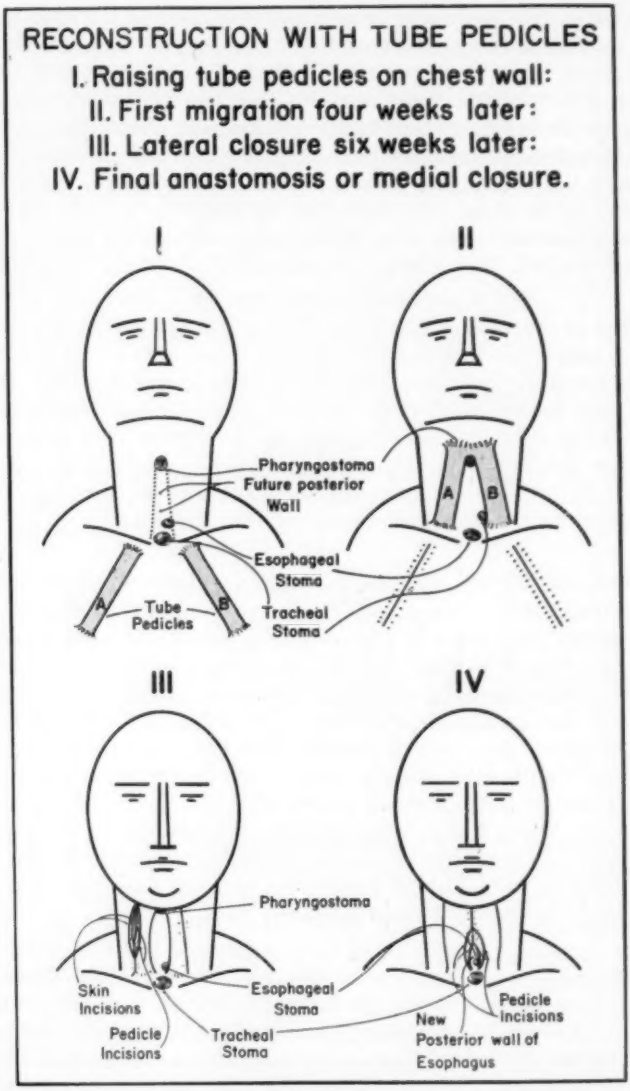
RECONSTRUCTION WITH TUBE PEDICLES
I. Raising tube pedicles on chest wall:
II. First migration four weeks later:
III. Lateral closure six weeks later:
IV. Final anastomosis or medial closure.
I
II
Pharyngostoma
Future posterior Wall
Esophageal Stoma
Tracheal Stoma
A
Tube Pedicles
B
A
B
III
IV
Pharyngostoma
Skin Incisions
Pedicle Incisions
Esophageal Stoma
Tracheal Stoma
New Posterior wall of Esophagus
Pedicle Incisions

Fig. 3

this method was employed, and these patients became dependent upon feeding tubes for the rest of their lives.

As indicated above, most of these patients had maximum neck surgery which usually means that they have had a larynx, cervical esophagus, thyroid gland and parathyroid glands and contents of both lateral necks removed (see Fig. 4). Often it

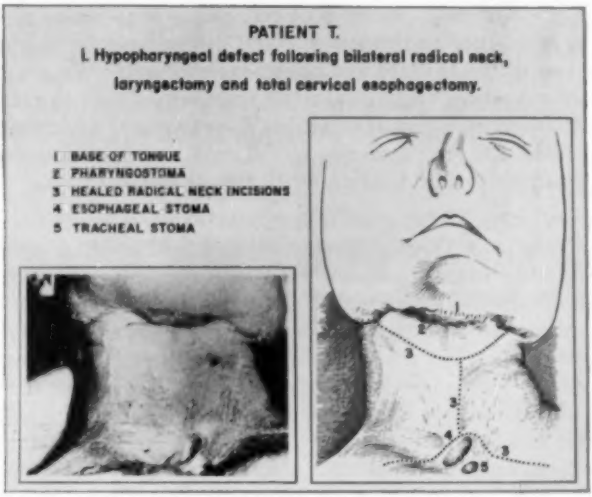
PATIENT T.
I. Hypopharyngeal defect following bilateral radical neck, laryngectomy and total cervical esophagectomy.
1 BASE OF TONGUE
2 PHARYNGOSTOMA
3 HEALED RADICAL NECK INCISIONS
4 ESOPHAGEAL STOMA
5 TRACHEAL STOMA

Fig. 4

was necessary to remove a large segment of skin in order to encompass the tumor. In addition, some patients had had heavy radiation to the neck at some time or another, and in these latter cases metabolism usually is controlled by the use of replacement therapy; that is, large doses of vitamin D, calcium, and thyroid extract. All these factors present complications in plastic surgery.

Often the pharyngostoma itself complicates the wound healing of the tube pedicles, because it is a likely source of infection (see Fig. 4). Great attention to wound care by the nursing staff, the resident, and by the attending staff is neces-

sary during the period when tubes are being migrated from the chest to the neck. In several cases we intentionally closed the pharyngostoma prior to migrating the tube pedicles to remove this problem of contamination when the pedicles were transferred. Once the tubes were migrated and an adequate blood supply was established, the pharyngostoma was opened. Frequently these patients are elderly, and since they are in an hypothyroid state, primary wound healing is very slow.

It is the clinical experience of our group that tissue resistance in these tube pedicles is low and directly related perhaps to the loss of the thyroid and parathyroid glands. We are sufficiently concerned to have started research in rats to clarify this situation. Few conclusions can be drawn to date, although we have done thyroidectomies on 50 rats and have compared their primary wound healing with that of normal rats.

We feel certain that even though adequate replacements for thyroid and parathyroid hormones are instituted, a certain percentage of wounds demonstrates a lower resistance to infection and primary healing. The two pedicle grafts often appear healthy for as long as seven days and then take on a localized gangrene about the suture lines. We are most interested in studying this phase of wound healing in these particular problems. It has been our experience that before a migration is made, and before the actual transplantation is undertaken, the surgeon must be certain of the circulation in the flap. Any simple method whereby the circulation in the pedicle flaps can be occluded gradually might be helpful and could save the patient and surgeon the chagrin of losing all or part of the flap after several operations have been performed.[25] Necrosis of any sizable amount can be most depressing to these patients and creates a difficult situation. If we encounter circulatory distress in the flap it is our policy to delay the separation by at least a week. For this reason, we have no standardized time for migrating the flaps. We try to make migrations approximately three weeks apart once we start this type of reconstruction; but we are frequently delayed because of circulatory distress. We have been successful in using this double tube method in several different types of cases.

We fashion bilateral or single, double tube pedicles on the

anterior chest wall and at three-week intervals migrate them to the defect in the neck for reconstruction. Sometimes it is possible to use just a single double-tube pedicle, particularly in cases where just one side of the neck is badly scarred. Normally the use of the two pedicles is more satisfactory, for we can effect reconstruction so that the patient can swallow and, in time, develop an intelligible esophageal voice.

The Wire Mesh and Skin Graft is a technique used frequently by some surgeons.[22,26,27,28] Primary reconstruction of the esophagus at the time of excisional surgery has been effected by bridging the gap between the proximal and distal ends of the cut esophagus with a wire mesh tantalum or stainless steel mesh which is prepared at the time to fit the defect. A polyethylene tube has been used by the British.[29,30] The wire mesh is rolled to form a cone resembling the hypopharynx and cervical esophagus. Then the mesh is covered by a thin split thickness skin graft taken from the thigh or abdomen with a Reese or Padgett's dermatome. The skin is sutured to this wire mesh in such a fashion that the epidermal side of the skin graft faces the lumen of the esophagus; the wire mesh acts as a support for the skin graft. The ends of the tantalum cone covered by the graft are sutured to the proximal and distal ends of the esophagus and the cone is placed into the neck. The vertical cut ends of the skin graft should be in immediate contact with the prevertebral fascia; this helps in preventing the formation of fistulae. The method was described originally (1949) by Rob and Bateman and is referred to in literature as the one-stage tantalum mesh method for reconstruction of an excised cervical esophagus.[31] These grafts heal very nicely, and there are numerous reports in the literature of rapid and complete functional rehabilitation of the esophagus. The tantalum or stainless steel mesh is removed as soon as the epitheliazation is completed; usually this is within four to six weeks. It is possible to view the area by the use of a hypopharyngeal mirror; a direct esophagoscopy may be performed if the surgeon is not certain that anastomosis is complete. The mesh is easy to remove by using strong alligator forceps and gently teasing it away from the new esophageal lumen. It is then crushed and removed endoscopically. At this stage the patient is swallowing. Swallowing begins in ten to twelve days, usu-

ally depending upon the appearance of the external healing of the neck. Closure over the mesh is similar to any closure, except one flap should be longer so that the suture line is lateral to the center of the wire mesh skin graft cone.

We used this method in two cases but did not find it as satisfactory as other surgeons reported. In one case the mesh appeared to act as a rigid foreign body causing necrosis by sheer pressure. This necrosis led to a severe localized infection and cellulitis; it was necessary to transfer a chest flap to the area to effect a closure. Edgerton reported this same objection in 1952 and Conley in 1953.[27,28]

In a second case, residual or recurrent disease became apparent within six months after the graft had healed and the patient had established function. It was our feeling that, in this particular case, perhaps the residual or recurrent disease near the prevertebral fascia had been masked or buried by this rather bulky reconstruction which overlay the site of the disease. This type of recurrence with the wire mesh method was reported by Rob and Bateman in their original three cases;[21] furthermore, we have seen cases where residual disease became evident after a pharyngostoma and esophagostoma were formed and before reconstruction was started. In one of these cases we were able to control the residual disease by performing a radical neck dissection on the opposite side. We felt that the residual disease was diagnosed much earlier than had we covered the area with a wire mesh cone and skin graft. In recent literature VonDielin has taken exception to this method and stated emphatically that one should not worry about recurrences since the return of function and appearance means much more to the individual patient.[32] He feels that primary reconstruction should be attempted as soon as it is at all feasible. Our group readily admits that some patients do become very depressed. Often there is a genuine problem of management when patients must ride through the multiple stages of reconstruction. For this reason we have tried to discover, with the help of the animal laboratory, why some of the multiple procedures fail. We have tried to evaluate each individual problem as it presented itself and have found two additional techniques which have been successful in speeding

up the reconstruction period in the cases to which they were applied. These new methods will be discussed next.

OUR INNOVATIONS.

The greatest objection to the use of the bilateral double-tube method is that it is a five to ten stage procedure at best and when finally completed the neck is a maze of suture lines.[23] It

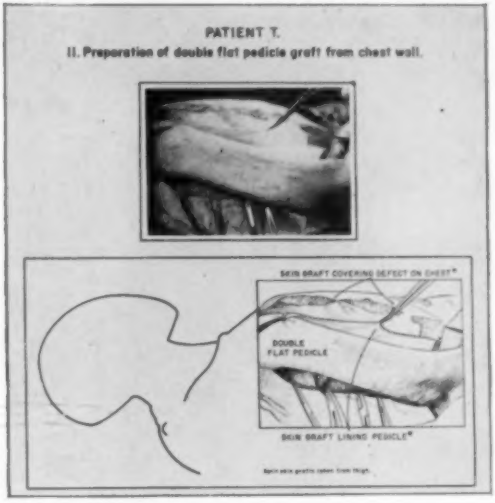
PATIENT T.
II. Preparation of double flat pedicle graft from chest wall.
SKIN GRAFT COVERING DEFECT ON CHEST
DOUBLE FLAT PEDICLE
SKIN GRAFT LINING PEDICLE

Fig. 5

is then possible, during healing, for these suture lines to contract and infringe upon the diameter of the reconstructed esophageal lumen. In addition, it always seemed necessary for us to raise two tube pedicles, thus doubling the operative morbidity and complications.

In an attempt to simplify these stages, we felt that a single broad double pedicle might be more satisfactory than a bilateral pedicle brought to the midline and then anastamosed. It seemed reasonable to use a flat pedicle rather than one tubed or circular, for it would mold to the neck and lend itself to

better and easier reconstruction. In our first case we raised a very wide (4 x 16 inches) double pedicle graft on the lateral chest wall taking care to use the main superficial vessels of the chest wall to nourish the pedicle. The pedicle was raised under local anesthesia and two full drum split thickness grafts were faced back to back (epidermal side to epidermal side). These skin grafts were placed under the raised tube pedicle in

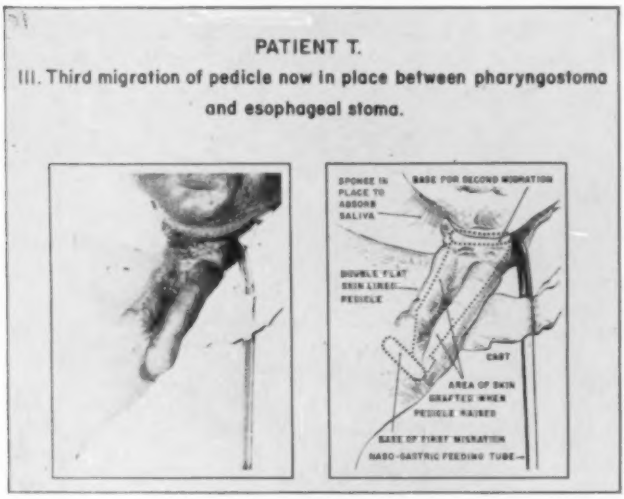
PATIENT T.
III. Third migration of pedicle now in place between pharyngostoma and esophageal stoma.
SPONGE IN PLACE TO ABSORB SALIVA
BASE FOR SECOND MIGRATION
DOUBLE FLAT SKIN LINED PEDICLE
CAST
AREA OF SKIN GRAFTED WHEN PEDICLE RAISED
BASE OF FIRST MIGRATION
NASO-GASTRIC FEEDING TUBE

Fig. 6

such a fashion that one closed the defect on the chest wall and the other served to close the raw surface on the elevated flat double tube pedicle (see Figs. 5 and 6). This procedure effected healing in a surprisingly short time. After gradual occlusion of the distal end, and after a good blood supply had been established, the tube was migrated to a point lateral and inferior to the tracheotomy opening. In the next stage it was moved to a position anterior to the pharyngostoma. At this time, the patient was placed in a cast to decrease the possibility of insult to the anastomosis of the tube to the submental area. The graft was shortened in the next stage and placed

in the neck so that its distal end was just below the esophagostoma. The lateral ends of the flat lined pedicle were sutured to the skin in layers on either side as in the bilateral double pedicle (see Fig. 3-4). This method of reconstruction proved very successful.

In another instance, where the excisional surgery included not only the larynx and cervical esophagus but also the posterior third of the tongue, the defect was more hypopharyngeal,

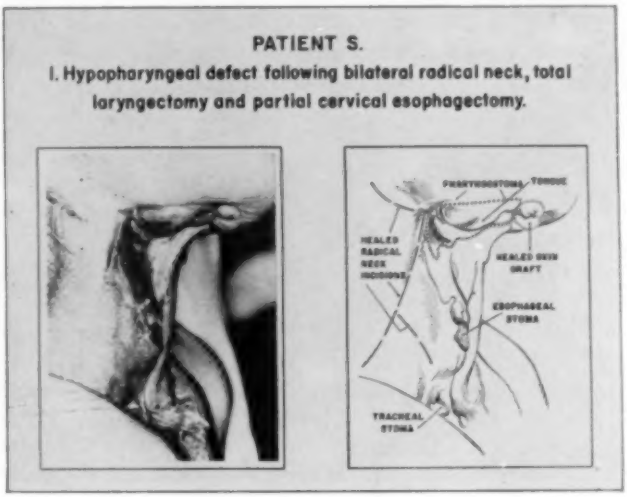

Fig. 7

and the center neck was markedly scarred. In this case a different approach was used. The original lesion in the hypopharynx was most extensive. In removing the posterior one-third of the tongue both hypoglossal nerves were severed. The remaining part of the tongue was used to fill the defect in the submental space forming a large pharyngostoma (see Fig. 8). A split thickness graft was used to help close the skin to the mucous membrane where it had been necessary to excise widely to remove skin involved with recurrent disease (see Fig. 8). Our biggest problem was to find tissue to form

the anterior wall of the hypopharyngeal defect. In this particular case we could have raised a large double-tube pedicle. The skin on the lateral neck was soft and pliable and could be used for the posterior wall. At this point we conceived the idea of using the base of the tongue (in reality the middle third, since a posterior one-third had already been removed). The tongue mobilized very well; it was already functionless and no problem to the patient. It was possible, by pulling, to

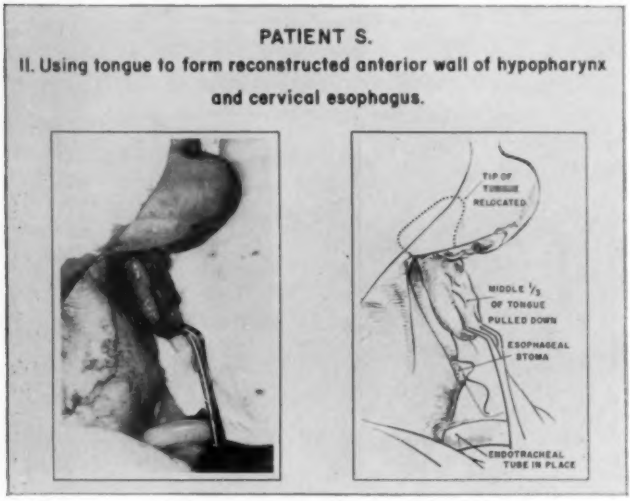
PATIENT S.
II. Using tongue to form reconstructed anterior wall of hypopharynx and cervical esophagus.
TIP OF TONGUE RELOCATED
MIDDLE 1/3 OF TONGUE PULLED DOWN
ESOPHAGEAL STOMA
ENDOTRACHEAL TUBE IN PLACE

Fig. 8

bring the middle third of the tongue down as low as the esophagostoma (see Figs. 7 and 8). This was done by reducing the thickness of the mucosa and mobilizing both sides of the floor of the mouth (see Figs. 7 and 8). The blood supply was excellent at all times. The anterior wall of the reconstructed defect was formed by using a modified "Z" plasty, and the scarred bilateral neck flaps were transferred to cover the entire defect (see Fig. 9). Although a portion of these external flaps sloughed out postoperatively, it was evident from the beginning that the inside layers were going to heal. This, I believe,

was due to the marked vascularity of the tongue tissue used to form the new anterior wall of the esophagus. It was a matter of seven to ten days before epithelization of the sloughed area external to the tongue began. The patient was able to swallow about three weeks after this one-stage reconstruction. By utilizing the tongue tissue I feel we saved the patient four to five months of intermittent hospitalization and several operative procedures.

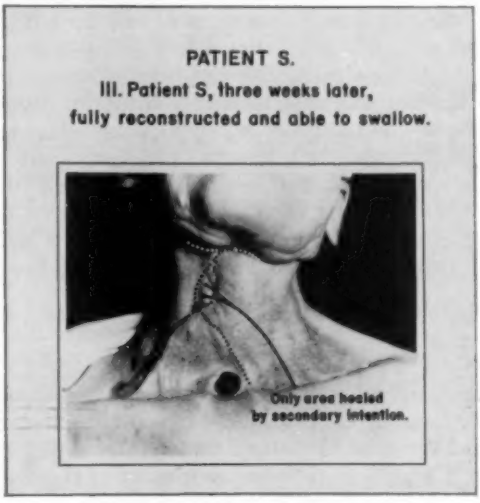
PATIENT S.
III. Patient S, three weeks later, fully reconstructed and able to swallow.
Only area healed by secondary intention.

Fig. 9

EXPERIMENTAL STUDIES.

Animal surgery already has contributed somewhat towards the advancement of esophageal and tracheal reconstruction work.[34,35] At present we are trying to grow tracheal homographs of dogs in their abdomens. We are trying to grow them in their peritoneal cavities and are trying to cover the inner surface of the tracheal homographs with intestinal mucosa. Our thought is that if these homographs grow we will transplant them to the neck by multiple stages in order to build a better esophagus. We have experimented considerably with

tantalum and wire mesh together with polyethylene tubes and skin grafts. We feel that the use of soft sponge rubber as a splint is far superior to the rigid splints. It has been mentioned that rigid splints may cause pressure necrosis. We believe we have solved one of the more difficult problems found in reconstruction work in dogs. We have constructed a tracheotomy tube patterned after a tracheotomy tube which is inserted into horses when they are racing.

As far as we know this is the first time this type of tube has ever been used in dogs. We were able to keep a dog alive for two months with this tube. Previously, most of our dogs died from two to three weeks with the conventional type tube or when the trachea was exteriorized to the skin. Other investigators have successfully used a polyethylene tube as a tracheal implant. In four months a fibrous lining grew into the tube.[36] Our enthusiasm for animal reconstruction work in this area becomes more vigorous each year as progress is noted. We are hoping that it will be possible to reconstruct a larynx. Certainly the reconstruction of half a larynx has been successful.[37]

SUMMARY.

In reviewing the literature it becomes apparent that radical surgery for the control of advanced hypopharyngeal, laryngeal, and cervical esophageal malignancies is not a new method of treatment. Large defects in the continuity of the pharynx and the esophagus have been a necessary result of this type of surgery since the first operations in the Nineteenth Century. Little attention was given to reconstruction problems by early surgeons and their morbidity and mortality rate was prohibitive. Excisional surgery was inadequate, and recurrent or residual disease eventually took its toll from the few apparent successes.

Between 1920 and 1926 more attention was given to the reconstruction and rehabilitation phase of head and neck surgery. The number of complete cures remained low, because surgery was still inadequate. In recent times excisional surgery has become a primary method of treatment for advanced malignancies in this region. Larger and more complicated de-

fects are resulting, and reconstruction is becoming increasingly complicated. Since reconstruction of these defects is essential and highly individualized, every successful technique should be reported.

CONCLUSIONS.

1. Four reconstruction methods used successfully by others have been tried by our group. These procedures are described and compared. Our experiences and observations with each method are discussed.

2. Two additional methods have been used recently by our group with apparent success. These procedures are described and discussed.

3. Experimental work in animals (dogs and rats especially) has been started. This work can be exceedingly helpful in studying the problems of reconstruction and in applying any new techniques.

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IDIOPATHIC HEMOTYMPANUM.

REVIEW OF THE LITERATURE AND REPORT OF THREE CASES.*

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Judging from the paucity of reports in medical literature, idiopathic hemotympanum is uncommon. The condition is mentioned in only one[1] of the current textbooks that we have reviewed. Since 1951, three cases have been treated in this hospital: the first by simple mastoidectomy; the second by radical mastoidectomy; and the third by simple mastoidectomy, followed by two attempts to produce a permanent perforation in the tympanic membrane.

In 1952, Menck-Thygesen[2] reviewed the literature and listed 11 cases, including two of his own. The diagnosis on two of these[3] was not definitely established, myringotomy was not performed, and the character of the fluid in the middle ear of either of these two cases was not known. Since the condition completely cleared up following adenotonsillectomy, serous otitis media appears to be a more plausible diagnosis. In this latter condition it is not uncommon for the tympanic membrane to present a dark bluish cast, similar to the color seen in hemotympanum.[4] Baron speaks of this color as "gun-metal blue".[5] We, therefore, feel that these two cases should not be classified as Idiopathic Hemotympanum.

One case of blue eardrum, discussed in considerable detail by Johnston,[6] in his paper on idiopathic blue eardrum, was described by Vior.[7] The discoloration was limited to the posterior inferior quadrant of the drum membrane, a definite pulsation was visible, and there was a pronounced bulge which

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Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication, November 23, 1955.

increased in the blue area when the homolateral jugular vein was compressed. Vior concluded that his case was one of jugular bulb protrusion. This is another case that should not be listed under the title, Idiopathic Hemotympanum.

Johnston,[6] in 1953, reported two cases. After eliminating the three cases previously mentioned (O'Donnell and Vior) as cases that should not be classified as Idiopathic Hemotympanum, the total number of cases previously reported is 11. The three cases herein reported bring the total up to 14 at the time of this writing.

CLINICAL PICTURE.

In the cases reported both male and female have been affected, and the ages range from eight to 60. In no case have both ears been affected. The patients have come for treatment because of pain, discharge or hearing loss. If pain is present, it is usually intermittent and suggests Eustachian tube closure.

The pain in some cases has been associated with acute otitis media. The hearing loss is conductive, the only unusual aspect being that in some cases it has been much greater than is usually seen with fluid in the middle ear. If discharge has occurred, it may be purulent (from acute suppuration), or reddish brown (chocolate colored). One case[2] developed immediately following acute suppurative otitis media.

Examination of the patient may reveal nothing of significance other than the otologic findings on the affected side. The tympanic membrane is intact (if discharge has not occurred recently) and might suggest serous otitis media on first glance, because of the bluish color; but it can be seen that it is not the gun-metal blue of serous otitis, but rather a definite bluish-black suggesting hemotympanum. In a number of the cases the tympanic membrane has appeared to be retracted despite a patent Eustachian tube, and did not move perceptively with inflation. The Eustachian tube has been open in the majority of cases, resulting in the conclusion that Eustachian tube closure probably is not a factor.[8] The most important finding from a standpoint of diagnosis is the character of the fluid found in the middle ear. All reported cases subjected to myr-

ingotomy have shown a fluid that appeared to have come from a liquified hematoma. This is reddish-brown, chocolate-like, and often moderately viscous. Detailed laboratory examinations[2,8] have shown this fluid to be sterile blood, or blood products. Roentgenograms of the mastoid usually show a pneumatized bone with clouding of the air cells, fuzziness of the intercellular septa, and may show bone destruction. Some cases have had a sclerotic mastoid.

DIFFERENTIAL DIAGNOSIS.

Johnston[6] goes into detail on the various conditions which may cause a "blue eardrum". These include hemotympanum due to head injury, barotrauma, or hemorrhagic diathesis; vascular anomalies, such as varices of the middle ear or protrusion of the jugular bulb; and conditions resulting from obstruction of the Eustachian tube such as the rupture of a hemorrhagic bleb in acute otitis media, or serous otitis. Serous otitis media is seen more often than any of the other conditions mentioned above, but if the tympanic membrane is blue, it is a gray-blue or gun-metal blue, and not bluish-black. Because of the confusion that is fostered by the use of the term "blue eardrum"[2,6] as synonymous with idiopathic hemotympanum, we suggest that the former term be dropped. We agree with Johnston, who mentioned that the most important diagnostic finding was the chocolate-like fluid found on myringotomy.

TREATMENT AND RESULTS.

When first seen most cases have been treated similarly to serous otitis media, with myringotomies and Eustachian tube inflation. This has never resulted in cure of the condition as far as is known. Menck-Thygesen[2] noted in his cases that repeated paracentesis and inflation led to progressively less and less improvement in hearing, and finally no improvement. He also noted that the tympanic membrane became anesthetic in time. These findings were not present in our cases.

Of the 11 cases of idiopathic hemotympanum reported, one[9] received no treatment, and five[2,6,10,11] were treated by repeated inflations and myringotomies with only fair to disappointing results. Tubal obstruction was felt to be a factor in one case;

obstruction was not commented upon in two; and in two the Eustachian tube was patent.

The treatment and results were not stated in one[1] case.

The remaining four cases were subjected to simple mastoidectomy. Ranger[8] operated on a child and a young adult. Neither had tubal obstruction as a factor, and prior to mastoidectomy repeated myringotomies and inflations were carried out, but without improvement. Mastoid X-rays showed evidence of disease other than simple cloudiness due to fluid, and at surgery the mastoids were filled with the same chocolate-like viscous material that had been obtained by myringotomy. There was no bone destruction. Analysis of the fluid showed no bacterial growth, and that it was essentially altered blood. Microscopically there were heavy cholesterol crystal deposits, multinucleated giant cells, and "cholesteatomatous tissue" without squamous epithelium. Both patients had a normal tympanic membrane post-operatively, and the child's hearing was markedly improved—an 11 db loss in the speech range.

Menck-Thygesen[2] operated on a ten-year-old boy who developed idiopathic hemotympanum following an acute suppurative otitis media. There was no persistent tubal closure in this case after the condition developed, although it was assumed that tubal closure was, in part, responsible for the preceding acute otitis media. The "viscous brownish-red fluid" was sterile, and was shown to be a product of blood. Roentgenograms showed a well pneumatized mastoid with blurred cell walls and some bone destruction. At surgery the mastoid was filled with a fluid and a type of granulation tissue similar to that described in Ranger's cases. Post-operatively the hearing and tympanic membrane returned to normal.

In 1953 Johnston[6] reported a case of a boy, first seen at the age of eight, with an acute suppurative otitis media, one month following an adenotonsillectomy. On penicillin therapy he made an uneventful recovery and was seen periodically thereafter. The tympanic membrane was normal, and the Eustachian tube open. A 35 db conductive hearing loss, which did not change with tubal inflation, developed. One year following

Fig. 1. Low power view.

the acute episode the drum membrane took on the appearance of an unresolved blood clot. During the following four years the color of the tympanic membrane and the hearing loss remained the same, and there were three or four episodes of spontaneous otorrhea of thick reddish-brown material, all of which subsided with penicillin therapy. Roentgenograms demonstrated a well-pneumatized mastoid with diffusely increased density, and a fuzzy altered trabecular pattern. Five years after the initial episode a simple mastoidectomy was per-

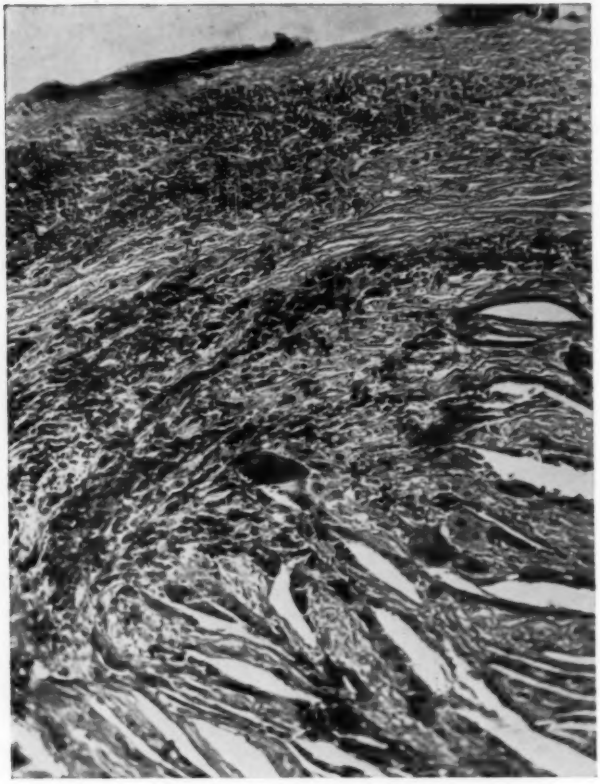

Fig. 2. High power view.

formed. All cells were filled with sterile, thick reddish-brown material, and there was a suggestion of pressure necrosis of bone in the antrum. Tissue removed at surgery showed fibroblastic proliferation, chronic granulation tissue with cholesterol crystals and foreign body giant cells. Six weeks following surgery there was a marked hearing improvement, although there was still a minute amount of fluid in the middle ear, and a reappearance of the blue-black coloring of the eardrum.

The reason for performing simple mastoidectomy in these

cases are three: first, it appears that there is some disease process going on in the mastoid cells themselves, and mastoidectomy is a logical approach to a cure, even though the cause is not known. Second, whatever the disease, the mastoid space seems to be acting as a reservoir for fluid, and its obliteration is logical as an approach to cure. Finally, there is the possibility that there is some minute bleeding area somewhere in the mastoid, and, although it may not be seen, an obliteration of the mastoid cavity might bring a halt to the disease or manifestations of the disease.

Of the four cases subjected to simple mastoidectomy, three were cured of their disease process. The fourth case[6] had a marked hearing improvement, but persisted in forming fluid, and maintained the blue-black eardrum.

ETIOLOGY.

Nothing is known of the cause of this condition. Most writers who have discussed it have come to the conclusion that it is best called idiopathic. It seems likely that somewhere in the mastoid, tympanum, or Eustachian tube there is a small area that bleeds. Surgery has not uncovered the source thus far.

Pathologically all cases coming to mastoidectomy, including the three cases presented herein, have shown the same general nonspecific picture: fibrous connective tissue containing numerous small blood vessels, chronic inflammatory cells, cholesterol clefts, and a variable number of foreign body giant cells (see Figs. 1 and 2). Cultures of the viscous reddish-brown fluid that is found have been negative.

CASE REPORTS.

Case 1: A 17-year-old white boy was first seen at this hospital in March, 1951, because of progressive hearing loss and recurrent attacks of drainage from his right ear. An adenotonsillectomy had been performed in 1941. He first had ear trouble in 1948, and since that time had five attacks of what sounded like uncomplicated acute suppurative otitis media, all but one attack being in the right ear. In 1949 a readenoidectomy was done, followed by radium irradiation to the nasopharynx. At that time the record shows that both tympanic membranes were intact; no mention was made of discoloration. He continued to have a slowly progressive hearing loss and attacks of otitis media. His general health was good.

On examination the nasopharynx showed some regrowth of adenoids, especially in the fossae of Rosenmueller. There was a thin, reddish-

brown discharge issuing from a central perforation of the right eardrum, the result of an acute otitis media. After two weeks the discharge had ceased (penicillin was given during part of this time), and the drum membrane is recorded as being black and retracted.

In April a re-adenoidectomy was done, but this had no effect on the general picture. There was an average loss of 33 db in the speech-hearing range.

Examination in July revealed no change in the hearing. The Eustachian tube was inflated with ease. The right eardrum was described as shiny and black, with a tit-like projection postero-inferiorly and a bleb-like projection at the pars flaccida. Roentgenograms revealed a well pneumatized mastoid with diffuse clouding. Simple mastoidectomy was performed* revealing a mastoid filled with slightly viscous reddish-brown fluid, and dark granulation tissue. The antrum was free of this granulation tissue.

Histologic examination of this tissue revealed a fibrous connective tissue in which there were numerous cholesterol clefts surrounded by multinucleated giant cells. There were scattered areas of hemorrhage and chronic inflammatory cells.

The post-operative course was uneventful, and examination of the ear 15 days later revealed a persistence of the black coloring of the tympanic membrane.

Follow-up examination performed by Capt. W. R. Green at Fitzsimmons Army Hospital, March 21, 1955, showed the tympanic membranes to be essentially normal and audiogram showed no hearing loss.

Case 2: A 20-year-old man of Japanese descent was first seen at this hospital in January, 1953, because of a two-week episode of "bloody discharge" from his left ear three months previously.

He had first noted hearing loss in his left ear seven years previously, and believed he had had some discharge from the ear three years prior to that. He had intermittent earaches for three years, and tinnitus for two years. Three months prior to hospital admission he developed pain in his ear which was relieved by spontaneous discharge of a bloody material.

Examination revealed the nasopharynx to be relatively free of adenoid tissue. The right ear was normal. The left external auditory canal was partially filled with dark brown crusted material which appeared to be old blood. Upon removing this the tympanic membrane was seen to be blue-black and intact. The Eustachian tube was inflated with ease, and no fluid could be heard, nor could bubbles be seen. Audiogram demonstrated a relatively flat conductive hearing loss of 60 db. Roentgenograms showed a densely clouded mastoid in which cellular detail could not be made out. There was a suggestion of bone erosion in the squamosal area.

On Feb. 10, 1953, a mastoidectomy was performed. A bluish-black material of soft consistency filled the entire mastoid cavity, extending into zygomatic and squamosal cells. The bone of the cellular mastoid was softer than normal. When it was found that this same bluish-black material filled the attic and tympanum, a radical procedure was done.

Microscopic examination of this tissue revealed many cholesterol clefts, surrounded by rather dense fibrous tissue infiltrated with lymphocytes and containing numerous small endothelial lined blood vessels. Foreign body giant cells were also present.

* Surgery performed by Major Samuel Lee and Colonel Wendell A. Weller.

The post-operative course was smooth. The cavity was completely lined by squamous epithelium 12 weeks following surgery, and there had been a 24 db hearing improvement, to the 36 db level.

COMMENTS.

It is unlikely that any procedure short of radical mastoidectomy would have cured this patient, due to the large amount of abnormal tissue in the middle ear space. The hearing improvement following surgery was gratifying to the patient as well as to us.

Case 3: A 21-year-old white man, complaining of recurrent pain and deafness in his right ear, was admitted to this hospital in June, 1953. He stated that he had never been able to hear with his right ear, and as long as he could remember he would have four or five episodes of pain a year, lasting from a few days to a few weeks. Five years prior to this hospitalization he was told that he had a blue eardrum, and since that time has been told on a few occasions that he had dried blood in his ear canal. He did not remember any episode of frank discharge. Mild symptoms of vasomotor rhinitis had been present for years, but had not been particularly bothersome. He had been referred to this hospital because of a "dark fluid" draining from the right Eustachian tube area after attempted catheter inflation.

Examination revealed a moderate amount of lymphoid tissue over the walls of the nasopharynx and the fossae of Rosenmueller, but no obstruction to normal movement of the torus on swallowing. The left tympanic membrane was normal. The right tympanic membrane was intact, appeared to be moderately retracted, and was blue-black. There was no fluid level. Initial audiometric examination (pure tone and speech) revealed to 60 db conductive hearing loss in the right ear, and normal hearing in the left ear. Roentgenograms revealed a pneumatized but densely clouded right mastoid.

All attempts to inflate the right Eustachian tube were unsuccessful, and at no time was any fluid seen to escape from the tubal orifice as had been described prior to entry. Myringotomy resulted in discharge of a chocolate-like material, similar to fluid from a liquified hematoma. The fluid was under slight pressure, and a large quantity was removed by suction. Hearing improved to the 28-30 db level following almost every myringotomy and aspiration.

A diagnosis of idiopathic hemotympanum was made, but because of the report of the referring physician of abnormal discharge from the right Eustachian tube, a biopsy was taken from the right fossa of Rosenmueller. Histologic examination showed normal adenoid tissue.

On July 17, 1953, a simple mastoidectomy and wide myringotomy were performed. The mastoid was well pneumatized, and all cells with the exception of the immediate peri-antral cells and antrum were filled with a dark granulation-like tissue and chocolate-like fluid. Sections of the tissue removed revealed osseous and fibrous tissue, with an inflammatory infiltrate in the latter consisting of numerous lymphocytes and polymorphonuclear neutrophiles. There was a proliferation of fibroblasts, and many hemosiderin granules and cholesterol clefts were present. Foreign body giant cells were numerous. Aerobic and anaerobic cultures of the fluid and tissue removed failed to show any growth.

The post-operative course was uneventful, and ten days following

surgery the myringotomy wound was healed and the drum appeared blue-black. There was no hearing improvement, although the patient stated that the ear would occasionally "click and pop," at which time his hearing would improve momentarily. Eustachian tube inflation, however, was unsuccessful.

By the middle of September, although there had been no change in the hearing, there was a change in the eardrum. It took on a gun-metal blue color similar to that seen in serous otitis media, and the fluid became more serous, with much less coloring. It was felt that treatment, aimed primarily at opening the Eustachian tube, would perhaps cure the condition.

A re-adenoidectomy was done, followed in four weeks by a fractionated course of radiation in the region of the nasopharynx, delivering a total of 450 *r* in air to both right and left nasopharyngeal ports over a two-week period.

A month following the completion of X-ray therapy the hearing was at a 32 db level, although there was still serous fluid in the middle ear and a gun-metal blue eardum. No myringotomies had been done in more than a month; however, in the following month the recurrent otalgia returned, and the hearing dropped to a 50 db level.

Based on the experience with Case 2, at this point it was decided that radical mastoidectomy was indicated to rid the patient of his recurrent pain, which was more of a disability to him than the hearing loss. The establishment of a permanent perforation in the tympanic membrane was entertained as a more conservative procedure and one that could possibly result in better hearing than following a radical operation.

On Jan. 21, 1954, under general anesthesia the lower half of the tympanic membrane was turned up, together with the annulus and a few millimeters of skin of the external canal. The middle ear was normal, including the tubal area. A wedged-shaped section of tympanic membrane, including the annulus at the base, was removed, skeletonizing the tip of the manubrium. This represented approximately one quarter of the pars tensa, in the posterior inferior quadrant. Post-operatively a mild otitis media and a bothersome external otitis developed. Both subsided within a week. An audiogram done six days following surgery showed a 23 db hearing loss. Unfortunately this level was never reached again.

No attempt was made to maintain the opening of the perforation, for this was felt to be unnecessary. As healing took place the anterior and posterior edge of the perforation were seen to be on a different plane, the anterior edge being about one to two mm. medial to the posterior edge. There was very little discharge after the first two weeks. Four weeks following surgery the perforation had healed, and the hearing had dropped to 50 db. Again recurrent pain became a complaint, and the eardrum took on a gun-metal blue color. Serous fluid could be aspirated through a myringotomy incision.

On March 18 a further attempt at permanent perforation was made. No evidence remained of the operation just two months before. One third of the pars tensa was removed, the tympanic sulcus was curetted thoroughly, and a selvedge edge gauze pack was placed in the tympanum and external canal to maintain the opening. Wide spectrum antibiotics were prescribed in large doses to prevent a recurrence of otitis media and externa. One week following surgery the gauze pack was removed. The opening was large, the ear was dry, and the hearing was at a 28 db level. Within five days the opening had decreased greatly in size. To prevent closure a small polyethylene tube was placed through the opening into the middle ear and was left in place one month. During

this time there were no apparent reactions to the tube. There was no discharge, only mild intermittent pain, and the hearing remained near the 30 db level. When the tube was removed there was a small opening near the floor of the canal, without crusting. Within 24 hours the tympanic membrane had healed, and the hearing dropped to a 40 db level.

Further treatment in the form of a radical mastoidectomy was offered the patient, but was declined for the present.

COMMENTS.

Although this was a typical case of idiopathic hemotympanum, following simple mastoidectomy, it took on the appearance of serous otitis media. It is possible that both conditions existed, but that the serous otitis was masked by the hemotympanum, and that the simple mastoidectomy actually cured the latter. Persistent complete blockage of the Eustachian tube is not common in serous otitis, and the relationship of this to these two conditions is not clear.

Permanent perforation of the tympanic membrane has been used in some resistant cases of serous otitis media with good results,[12] and was suggested to the authors in this case. Immediately following this procedure the hearing was excellent, and, had the opening remained, a permanent improvement could have been expected. Considering the difficulty encountered in attempting to close some perforations associated with a quiescent chronic otorrhea, it is hard to explain why both attempts at permanent perforation failed here. This is particularly true in light of the fact that both were made marginally by removing a portion of the annulus tympanicus.

SUMMARY.

The literature on idiopathic hemotympanum is reviewed. A brief discussion of the clinical course, differential diagnosis, treatment and etiology is presented, along with a detailed description of three cases. The first case was cured by simple mastoidectomy. The second case was treated by radical mastoidectomy, resulting in a cure, but with a conductive hearing loss, less severe than before surgery. The third case was treated unsuccessfully by simple mastoidectomy and two attempts to produce a permanent tympanic perforation.

Since this manuscript was prepared, another case[13] of idio-

pathic hemotympanum has been reported. Radical mastoidectomy failed to reveal the etiology of the condition, and failed to relieve the patient's otalgia.

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INTERNATIONAL VOICE CONFERENCE.

Following the International Congress of Otolaryngology in Washington, D. C., next Spring, there will be an International Voice Conference (Laryngeal Research, Function and Therapy) in Chicago, Illinois, May 20-22, 1957. For Information address: Dr. Hans von Leden, 30 North Michigan, Chicago 2, Ill., U.S.A.

THE SIGNIFICANCE AND FATE OF THE MESENCHYME OF THE MIDDLE EAR.*

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In man, resorption of tympanal mesenchyme begins at the third fetal month and should, ideally, be complete by the eighth. In actuality, traces of this tissue may fairly often be present up to the fourth or fifth year, and a few children retain even larger amounts until the onset of puberty when, under the influence of hormones, any mesenchymal remnants tend to differentiate into adult fibrous tissue. The middle ear seems to be one of the last outposts where primitive mesenchyme as an organized tissue (syncytial reticulum) may be found in the developing individual. Pluripotential mesenchyme cells still exist, to be sure, in all the connective tissues and throughout the reticulo-endothelial system, but the masses of syncytium so characteristic of the early embryo are no longer elsewhere in evidence. That the ear should behave in this fashion is perhaps not so strange when we recall that the head of the young embryo consists largely of a mass of primitive mesenchyme containing a brain. The role of mesenchyme, so vast in embryogenesis as a whole, is, therefore, the more striking in the development of the head segment.

Failure of resorption of mesenchyme in the middle ear of man may quite possibly be a hereditary characteristic, and one conceivably associated with some subtle endocrinopathy or metabolic disturbance.[1] It has been pointed[1] out that this fail-

* Read in part at the meeting of the American Association of Anatomists, Philadelphia, Pa., April, 1955.

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† This research is being conducted in the Department of Histology of Creighton University under a grant from The National Institute of Health.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication, March 20, 1956.

ure may explain the well-known predisposition of certain families to otitis media and its complications. Every observant otologist and pediatrist has been struck by the high incidence of this disease in certain families as contrasted with its virtual absence in others. General familial hyporesistance to infection can hardly account for this phenomenon since members of such families are not necessarily afflicted by a predisposition to other infections. One is obliged, therefore, to assume that the middle ear constitutes in them a *locus minoris resistentiae* on the basis of some local anatomic peculiarity.

Dorothy Wolff[2] pointed out, as early as 1934, that infection and mesenchyme in the middle ear had a mutually unfavorable effect upon one another, infection stimulating differentiation of mesenchyme into scar tissue, and mesenchyme interfering with effectiveness of myringotomy in premature infants. (It is worth mentioning that this interference with drainage is by no means confined to premature infants but has been encountered, knowingly or unknowingly, by every experienced clinician in children of all ages up to puberty.)

The unfavorable effect of unresorbed mesenchyme on the course of otitis media through interference with drainage of exudate was further emphasized in 1936 by L. K. Guggenheim[1] in his study of the Marvin Jones collection (New York Postgraduate Hospital) of temporal bones from children dying of complications of ear infections. There was a striking incidence of large masses of tympanal mesenchyme infiltrated with inflammatory cells, with walled-off pus pockets throughout the tympanic cavity.

No doubt the deleterious effects of unresorbed mesenchyme upon the course of otitis media are more subtle and difficult of detection in this antibiotic age than formerly, and one would hardly be in a position to consider an unfavorable course of events as diagnostic of the persistence of mesenchyme. Wolff[2] mentions recovery of free blood on myringotomy as typical, due to the considerable vascularity of this tissue. Certainly this sign cannot be considered pathognomonic either, since recovery of free blood is also a well-known occurrence with fulminating infections due to Beta-hemolytic streptococci. A more suggestive sign is the experience, upon performing myringot-

omy, of a sensation such as one would get in cutting through sponge rubber.[3]

Another phenomenon observed in the ears of certain children independently by Hubert Peugnet[4] and one of us (P. G.) is a lacy, reticulated pattern resembling frost on the inner surface of the drum. The pattern has been noted to change with subsequent vicissitudes of some of these ears—additional infections, surgery, etc. Quite probably this finding is either due to primitive mesenchyme itself, or to what L. K. Guggenheim[5] called "reversible tympanal fibrosis" in children: "immature connective tissue deposited as a result of tubal obstruction from adenoid and mesenchyme, serous transudate or subclinical exudate—tissue which is capable of disappearing by mobilization of its cellular elements to wandering cells—with *restitutio ad integram* of the conduction apparatus."

It must be admitted, these limited observations notwithstanding, that no generally accurate method exists for clinically diagnosing presence of persistent tympanal mesenchyme; and if little is known about diagnosis, even less information exists regarding treatment. A single significant observation has been recorded by L. K. Guggenheim[1] in a small number of cases of children failing to show satisfactory improvement of hearing after direct adenoidectomy. These children were treated by the sole additional procedure of gentle dilatation of the tubal orifices with the Gyergyai olives. In a number of instances in which no adenoid remnants were found, the dilatation alone improved the hearing to normal. It was hypothesized that the procedure compressed some submucosal remnants of mesenchyme at the mouth of the tube, thereby presumably initiating a chain reaction which progressed distally, leading to resorption of the remainder of the persistent but unstable mesenchymal tissue. Such a reaction might well be mediated through liberation of an enzyme.

Although direct adenoidectomy has come into widespread use, the employment of the Gyergyai olives for tubal dilatation has not. Since it has been our practice to include this step in all cases in which adenoidectomy is preformed for hearing impairment or recurring otitis, we have not had occasion to repeat the observation of L. K. Guggenheim. Goodhill[6] has

suggested the experiment of performing unilateral tubal dilatation in cases of symmetric conduction impairment. This theoretically elegant idea presents one very practical difficulty. Whereas the failure of a few cases to achieve restitution of normal hearing after direct adenoidectomy *without any tubal dilatation* automatically selects a population in which one is entitled to make at least a presumptive diagnosis of unresorbed tubo-tympanal mesenchyme, this selectivity would not operate in so efficient a manner in the procedure suggested by Goodhill. The presumptive mesenchyme-bearing population would not be defined at the time of performance of the crucial procedure, and the requirement of initially symmetrical audiometric curves would drastically limit the sample from which this population would have to be drawn. Since the incidence of unresorbed mesenchyme in children of various ages has never been determined by routine temporal bone studies on general pediatric autopsy material (a study long overdue), accumulation of a fairly extensive series of satisfactory symmetric improvements would not necessarily prove the worthlessness of the unilateral dilatation; nor would its value be easy to estimate, given a few suggestive asymmetric results. Nevertheless, such a long term project should be carried out by someone with adequate clinical facilities in a large pediatric hearing clinic. This investigator should also undertake the tedious but highly necessary study of the incidence of unresorbed mesenchyme in children dying from *all* causes; for not only are otologic deaths today excessively rare, but even had an extensive statistical study of this kind been undertaken on otologic cases in the pre-chemotherapy era, an exaggerated picture of overall mesenchyme incidence would undoubtedly have resulted.

With regard to the safety of the tubal dilatation, Guild[7] has expressed some concern lest the pressure exerted by the olive damage the mucosa of the orifice, especially its delicate ciliary mechanism. It would not be difficult, nor in all probability harmful to biopsy the mucosa after performance of the dilatation; but it seems a scarcely permissible procedure to investigate an alleged possible injury to a patient by inflicting a certainly greater one. We do feel, after eight years' experience with the method, that it is a perfectly safe one. This impression is based in part upon an admittedly crude surgical esti-

mate of the effect upon the tissues of the quite moderate pressure employed, but more particularly upon the absence of any significantly deleterious effects recorded in our follow-up studies. One of us (P. G.)[8] reported rare instances of postoperative otitis media or serous effusion which responded readily to the usual treatments with no impairment whatever of the ultimate results. It is also worth mentioning that such complications will occasionally occur also after adenoidectomy *without* tubal dilatation.

In view of the widespread popularity of radiation treatment of adenoid remnants (and even of unremoved adenoids) which prevailed from 1941 until recently, it is rather surprising that no consideration has been given to the possible effects of ionizing radiation upon unresorbed tubo-tympanal mesenchyme. Conceivably such radiation could promote either resorption or differentiation into scar tissue. This problem is presently under experimental investigation in our laboratory.

It is readily apparent, in view of the paucity of both clinical and pathological information regarding unresorbed tubo-tympanal mesenchyme in man, that animal experimentation is needed. Moses Lurie,[9] of Harvard University, was the first to attempt this. Prior to 1936 he studied a strain of dwarf mice in which he found mesenchyme to be retained in the tympanic cavity indefinitely unless the animals were treated by injections of pituitary extract. L. K. Guggenheim[1] attempted to carry out similar experiments but with inconclusive results.

Stimulated by the work of these two men we have attempted to study the normal development of the middle ear of the white rat with reference especially to the cycle of growth and decay of the mesenchyme. It appears that this post natal study in the rat would correspond to an embryologic study in man because of the difference in the stage of the mesenchyme cycle of growth and resorption with respect to the event of birth. It seems likely, however, that much the same factors must be responsible for the behavior of this tissue in both rat and man. It is intended that the base-line established by this study shall be a standard against which to measure the effects of various chemical and physical agents which might experimentally influence the mesenchyme.

Realizing, too, that Lurie's dwarf mouse provided a most elegant experimental object, we attempted to procure such animals from the same sources. We found that the animals sent us exhibited a cycle of growth and regression of tympanal mesenchyme quite similar to that observed in our study of the white rat. Specimens from other widely scattered sources also revealed no instance of indefinite retention of mesenchyme. It seemed clear that Lurie had been so fortunate as to have obtained a rare mutant which is now either extinct or unavailable. The systematic study of the tympanal mesenchyme cycle of dwarf mice and their normal heterozygous littermates is presently being made and will be reported subsequently. The present report will be limited to results with the white rat.

Some comparisons between the ears of rat and man are useful for purposes of orientation in embryologic time. Ossification of the labyrinthine capsule of man first begins at the end of three months gestation, but is accomplished principally during the fifth month. The earliest deposition of calcium occurs in the matrix of the cartilage, most of which is subsequently removed by invading osteogenic buds. The remaining calcified matrix is converted into intrachondral bone when the swollen lacunae are invaded by osteogenic buds which proceed to lay down bone within them. Anson and Bast[10] speak of the cartilagenous otic capsule as being "almost completely converted into a bony capsule by the 202-mm. stage (23rd week)," but state that it consists at this stage of islands of intrachondral bone lying in large primitive marrow spaces. These spaces are only very slowly diminished by deposition of endochondral bone, a process which becomes greatly accelerated just before birth.

In the rat, as in man, the first evidence of impending bone formation is seen to be a swelling of the cartilage lacunae. This was first noted in the posterior lip of the internal auditory meatus of the rat at three days after birth. By five days extensive calcification has occurred on the medial side of the cochlear capsule, including the modiolus. The promontorial wall forming the lateral boundary of the second cochlear turn shows calcification (site of attachment of ligamentum spiralis). Centers are noted on the anterior lip of the internal

auditory meatus and on the medial aspect of the double ampulla of the vertical canals. The cochlear promontory and medial tympanic wall posterior to the oval window now show swelling of cartilage lacunae. By nine days, almost the entire cochlear capsule has been converted, and beginning changes are occurring in the various parts of the vestibular labyrinth. At this stage the bony labyrinth consists of a tenuous framework of intrachondral bone lying amidst enormous marrow spaces. In consequence, it has a most fragile appearance and is still capable of being sectioned by a sliding microtome without decalcification. From about ten days on, one can notice the earliest harbinger of endochondral bone formation in the form of thin lamellae of ossein matrix around the islands of intrachondral bone. From this time forth the capsule assumes a progressively more substantial appearance as the marrow spaces diminish in size. At 11 days the vestibular capsule is extensively converted. *A very rough estimate might equate a rat of five days with a human fetus of five months.*

With regard to the development of the cochlea, the classic work of Wada[11] states that the rat hears clearly on the 12th day of life, sometimes as early as the 10th or 11th day. (One instance was recorded in which hearing occurred on the ninth day.) The 12th day of the rat would, therefore, be comparable to the end of the sixth fetal month in man, when the organ of Corti is fully developed and physiologically competent. The week from the fifth to the 12th day in the rat thus *corresponds roughly to the sixth month of gestation in man.*

Wada's monograph[11] nowhere mentions mesenchyme, but we have had the opportunity of studying his original collection of slides at the Wistar Institute (being the second visitors to disturb them in the last quarter of a century). His sections are cut in a radial-vertical plane in order best to exhibit the cochlea. Ears were sectioned at three-day intervals from birth. An insufficient number of sections of each specimen was represented to tell the whole mesenchyme story, inasmuch as changes are occurring with a rapidity which demands specimens taken at daily intervals. The picture of development reconstructed from this series coincides very closely with that derived from our own.

Certain anatomic peculiarities of the rat are likely to confuse the otolaryngologist used to thinking in human terms. The nose adjoins the nasopharynx by a quite novel anatomic arrangement, which has been studied in detail by Kelemen.[12] The nasopharynx itself differs strikingly in configuration from man, being a long funnel-like structure which may be traced caudad in serial sections until it almost literally drops into the orifice of the larynx, the communication between it and the oral cavity being very small indeed.

No vestiges whatever of either tonsils or adenoids were seen in any of the animals studied by us, which were standard laboratory rats of the Sprague-Dawley and Holzman strains. It may well be that pharyngeal lymphoid tissue appears later in the animal's development than we followed it for our purposes. According to Greene,[13] rats, unlike other familiar laboratory animals, lack tonsils except as scattered lymphoid follicles. She makes no mention of adenoids.

TECHNICAL PROCEDURE AND HISTOLOGIC FINDINGS.

Rats were sacrificed at daily intervals from one to 27 days of age by transaortic injection of Zenker's solution or formalin under general anesthesia with intraperitoneal nembutal.* The internal jugular veins were severed in order to facilitate perfusion of the head. The animals were decapitated immediately postmortem, heads skinned, calvaria and brains removed, and the mandibles excised. The remains were further fixed by immersion in fixing fluid for 12 to 24 hours. Specimens from 11 days on were decalcified in three per cent nitric acid solution for 48 hours. Celloidin was used for embedding. Sections were hand cut by sliding microtome in a plane parallel to the hard palate. Those not decalcified were sectioned at 10μ thickness. The decalcified heads were sectioned at gradually increasing thicknesses, until, from 15 days on, all sections were of 20μ thickness. Sections were stained in rotating order with iron-hematoxylin, hematoxylin-eosin-azure, orcein, silver nitrate, and Mallory's connective tissue stain.

* Intravital fixation by injection under anesthesia was first employed by Siebenmann & Yoshii (1908), Metzner & Yoshii (1909) and Sato (1917), as mentioned by Wada (op. cit.) Yoshii used ether anesthesia and injected transaortically a solution consisting of: formol 10 per cent, 74 parts; Muellers fluid, 24 parts; glacial acetic acid, 2 parts.

In addition to the recording of descriptive data, an effort was made to study more precisely the growth and decay of the mesenchyme mass in relation to the overall growth of the potential tympanic cavity. Slides were projected onto standard white typing paper of known weight per unit area at 38x magnification, and the tracings cut out and weighed. From the paperweight and magnification data, actual areas on the slides were calculated. For purposes of convenience, the tympanum was divided into two regions, arbitrarily designated "Tubopromontorial" (TP) and "Stapedial foot-plate" (S). Insofar as possible, the TP region was represented by a section which

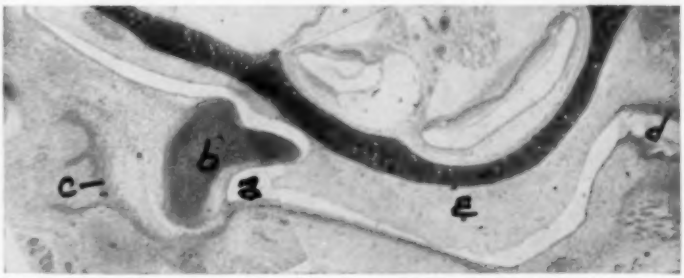


Fig. 1. Rat one day old. Composite photomicrograph showing entire extent of actual tympanic cavity at TP level.

a. Free tympanic lumen; b. Malleus; c. Drum membrane; d. Tympanic orifice of Eustachian tube; e. Anterior promontorial mesenchyme pad.

showed the full length of the Eustachian tube from nasopharynx to tympanum. The posterior boundary of this region, for purposes of quantitative study, was arbitrarily taken as the malleus. The S region was represented by a section showing the juncture of the anterior stapedial crus with the footplate. The S region thus lies cephalad to the TP region. Sections from the TP region of very young animals* show not only a corridor of free space lateral to the promontorial mesenchyme pad, but also a somewhat smaller chink extending just medial to the malleus and then posteriorly for almost the full length of the potential cavity (see Fig. 1). This posterior chink is evident only in sections caudal to the stapes

* We have studied specimens as early as one day prenatal. An embryologic series is now in preparation.

footplate, the S region being solidly packed with mesenchyme (see Fig. 2). Due to the arbitrary selection of levels for quantitative study, this extension of free space posterior to the malleus has not received systematic quantitative consideration.

Fig. 2. Rat at one day. Transverse section at stapes footplate level (S).

a. Comma of free space extending just medial to malleus but not further posterity at this more cephalic level (cf. Fig. 1); b. Tensor tympani muscle lying just lateral to cochlear promontory; c. Incus; d. Anterior crus of stapes; e. Nervus facialis; f. Stapedial footplate; g. Labyrinthine vestibule; h. Stapedial muscle; i. Stapedial artery.

Table I shows actual cross-section areas of mesenchyme in relation to areas for the whole potential tympanic cavity in the S region for animals of various ages.

Note that all specimens through the 11th day show a progressive increase in the size of the mesenchyme mass exactly paralleling the growth of the potential tympanic cavity at this level (S). After the 11th day, the mesenchyme begins to decline in the absolute amount while the S region of the potential cavity continues to grow very slowly. The amount of free

space gradually increases until by 20 days there is almost total elimination of mesenchyme.

TABLE I.

Stage (days)	Area of Potential Tympanic Cavity ($mm.^2$)	Area of Mesenchyme ($mm.^2$)	Area of Free Space ($mm.^2$)
1	3.4	3.4	0.0
2	4.6	4.6	0.0
3	6.6	6.6	0.0
5	6.6	6.6	0.0
7	6.9	6.9	0.0
9	7.2	7.2	0.0
11	7.5	7.5	0.0
12	7.8	6.9	0.9
13	8.2	5.7	2.5
15	10.2	3.3	6.9
16	10.2	1.2	9.0
17	9.7	1.3	8.4
19	10.0	1.2	8.8
20	10.3	1.2	9.1

Stapedial Footplate Region (S).

TABLE II.

Stage	Area of Potential Tympanic Cavity ($mm.^2$)	Area of Mesenchyme ($mm.^2$)	Area of Free Space ($mm.^2$)
1	2.5	0.6	1.9
2	3.4	2.5	0.9
3	4.0	2.5	1.5
5	4.0	2.8	1.2
7	8.5	3.9	4.6
9	9.5	3.4	6.1
11	17.0	5.4	11.6
13	20.0	0.4	19.6
15	21.5	0.0	21.5
16	22.0	0.0	22.0

Tubo-promontorial Region (TP).

Table II shows the same data for the TP region.

Here it will be noted that the trends are, in general, the same as in the S region, but certain differences are striking. The mesenchyme in the TP region does not fill the entire cavity on the first day, and figures for the mesenchyme and total cavity areas begin to diverge more rapidly than in the S region. As in the S region, the mesenchymal mass increases in absolute amount until the 11th day, when a maximum is reached. From the 11th to the 13th day there is a slow decline which becomes precipitous after the 13th day. The more ad-

vanced cavitation in the TP region is due primarily to the more rapid growth of this part of the potential cavity. It is obvious, too, that the resorption of mesenchyme proceeds more rapidly in the TP region, being complete at 15 days, whereas in the S region, remnants may linger up to 23 days. These changes are

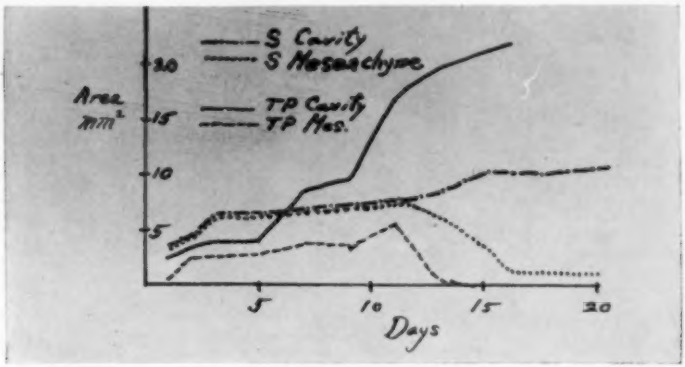


Fig. 3. Graphical representation of data in Tables I and II.

represented in the accompanying graph (see Fig. 3) and illustrated by the photomicrographs (see Figs. 1 and 2, and Figs. 4 to 10).

The foregoing study tells us nothing about the mechanism

Fig. 4. Rat at nine days. TP region.

a. Approach to the tympanic orifice of Eustachian tube (a′); b. Anterior promontorial mesenchyme polyp—Note greater density of ground substance, as indicated by more homogeneous, dark-staining appearance compared with c. mesenchyme of posterior part of tympanum which, as yet, shows no change preparatory to resorption; d. Malleus; e. Drum membrane.

Fig. 5. Rat at 12 days. TP region under higher magnification.

a. Tympanic orifice of Eustachian tube; b. Anterior promontorial polyp showing web-like structure with large holes, indicating impending dissolution.

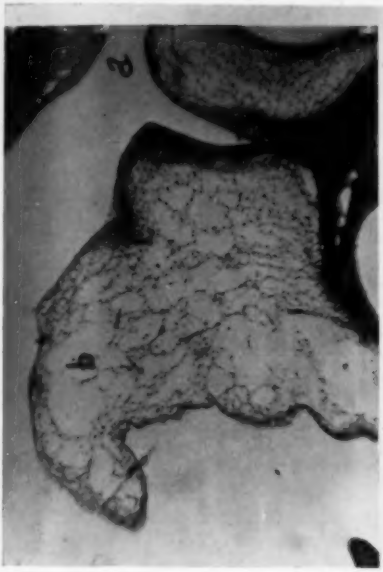
Fig. 6. Rat at 15 days. TP region. Note complete absence of mesenchyme at this level.

a. Tympanic orifice of Eustachian tube.

Fig. 7. Rat at 17 days. S region.

a. Tensor tympani muscle; b. Anterior crus of stapes; c. Posterior crus of stapes; d. Footplate of stapes; e. Stapedial artery; f. Facial nerve; g. Free tympanic lumen (g′, g″—holes in the mesenchyme around the stapes); h. Vestibule of the labyrinth; i. Stapedius muscle.

Note that various components of stapes appear to be composed of intrachondral bone.



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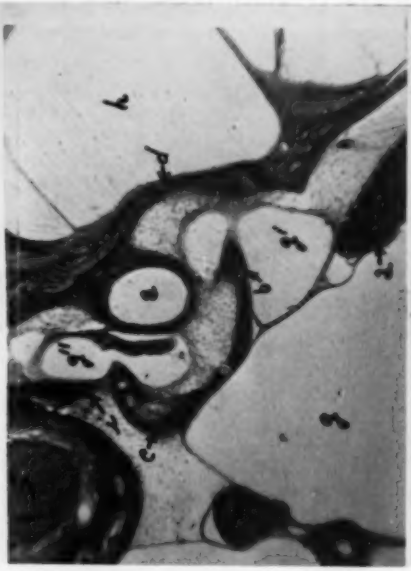

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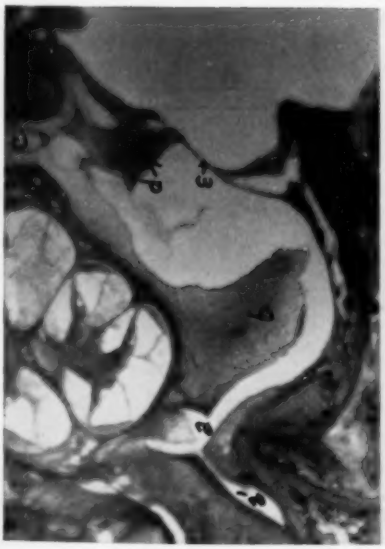

Fig. 4.

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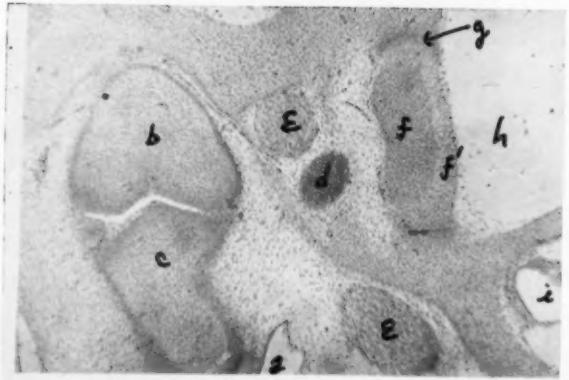

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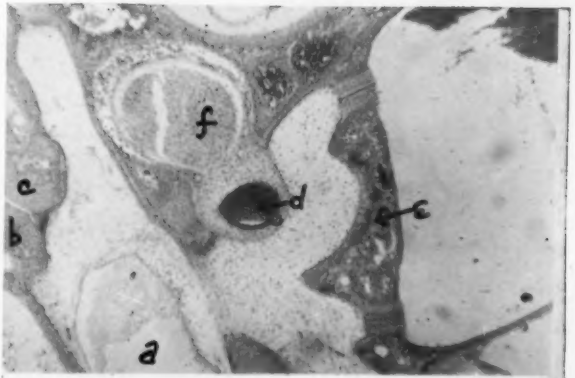

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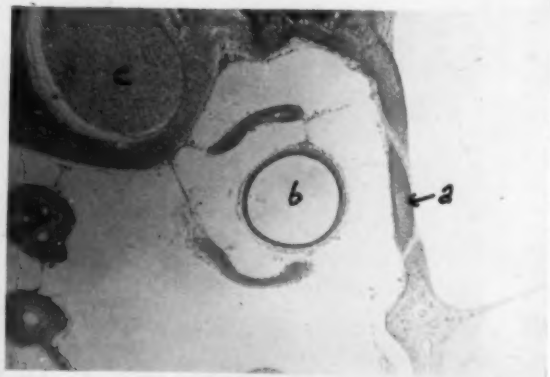

Fig. 10.

Fig. 8. Rat at one day. Wada collection,* vertical section, showing stapedial footplate.

a. Free tympanic lumen; b., c. Malleus, incus; d. Stapedial artery; e. Facial nerve; f. Part of the footplate derived from Reichert's ring —represented by darker-staining kidney-shaped area—flattened on its medial surface to accommodate itself to f.' part of the footplate derived from curtain of the oval window (labyrinthine capsule proper);[14] g. Anlage of annular ligament; h. Vestibule of labyrinth; i. cochlea.

Fig. 9. Rat at 12 days. Wada collection.* Footplate region. Vertical section.

a. Free tympanic lumen; b., c. Malleus, incus; d. Stapedial artery; e. Stapes footplate, now composed of intrachondral bone; f. Facial nerve.

Fig. 10. Rat 25 days. Wada collection,* vertical section, footplate region. No trace of mesenchyme remains in the stapedial region.

a. Footplate of stapes; b. Stapedial artery; c. Facial nerve.

* These photomicrographs were made at the Creighton University with permission of the Wistar Institute.

of dissolution of the tympanal mesenchyme mass—a most intriguing problem. A similar mesenchymal dissolution occurs in the formation of the perilymph chambers (at two months in man)[15] and of the subarachnoid space,[16] with the difference that the spaces vacated by the mesenchyme in these instances come to be occupied respectively by the homologous perilymph and cerebrospinal fluids, instead of by air as in the tympanic cavity. It may well be that a "liquefaction" occurs here also, the fluid being drained off through the Eustachian tube, or perhaps partly absorbed by adjacent lymphatics or capillaries.

The middle ear of the rat is one of the few locations where a mass of primitive mesenchyme is destined to disappear entirely after birth, and one which lends itself readily to observation. Otologists have termed the mechanism of this disappearance "resorption." Strictly speaking, the term "resorption" implies incorporation of the material "resorbed" into the structures of its environment. Undoubtedly some of the tympanic mesenchyme cells contribute to the elaboration of other structures in the potential tympanic cavity (especially those cells which are destined to become periosteum), but the vast majority simply disappear. "Dissolution" or "disintegration" would, therefore, seem better terms here than "resorption." The latter, however, is so entrenched in otologic literature as to be virtually invulnerable to such considerations. Obviously neither term could possibly be expected to epitomize so complex a process.

One cannot help being struck by a resemblance between the last remaining shreds of *moth-eaten* mesenchyme in the middle ear (see Fig. 7) and the trabeculae of the subarachnoid and perilymphatic spaces. Whereas these trabeculae have a most necessary supportive function where normally found, homologous remnants in the tympanic cavity represent an *arrest* of normal development and result in impaired mobility of the ossicular mechanism.

Schwarz[17] in 1931 spoke of "pneumatization" of the tympanic mesenchyme, claiming that this process begins earlier in the human fetus (three months) than previously supposed. The term "pneumatization" at once suggests to the otologic mind the mechanism of formation of the mastoid air cells.

Here, bone is actively reamed out by an invading epithelium in a manner which has been conceptualized by some as a normal analogue of carcinomatous invasion.

Bast and Anson[10] in their classic work retain this terminology, speaking of "pneumatization of the tympanic mesenchyme," and stating: "In a 22-week fetus the loose mucoid connective tissue gives way to the expanding tympanic epithelium."

TABLE III.

Age of Animal in Days	No. of Cells per 0.05 mm.²
1	6.7
2	7.0
3	6.7
5	5.0
7	2.0
9	1.7
11	1.3
12	1.7
13	0.7
15	3.0
16	0.0
17	0.7
18	5.3
19	0.0
20	0.7

Cell population density in the stapedial footplate region (S) as a function of age.

Dorothy Wolff,[2] on the other hand, mentions that the mesenchymal surfaces do not appear for the most part to be covered by an epithelium, and that scar tissue resulting from infection is distinguishable from mesenchyme partly by virtue of the fact that the former *is* covered by an epithelium.

Neither the cause nor the mechanism of dissolution of the tympanic mesenchyme are yet known, but some observations have been made on a cytologic level. In an effort to determine the extent to which changes in the volume of the mesenchymal mass were due to variations in cell concentration, the cell population density was studied in the TP and S regions at various ages, using a whipple micrometer disk. Three consecutive areas, each measuring 0.05 mm.,² were counted and averages taken. Table III shows these values in the S region.

It is evident that the cell concentration is at its highest level during the first three or four days of life, declining significantly on the fifth day, and precipitously by the seventh. With two isolated exceptions (15 and 18 day specimens),* all stages older than five days showed a marked reduction in cell population. Since the total absolute volume of mesenchyme increases up to a maximum on the eleventh day of life, there must be an increase in intercellular substance. The lessened cell popula-

TABLE IV.

Age of Animal in Days	No. of Cells per 0.05 mm.²
1	6.0
2	6.3
3	3.7
5	2.0
7	1.0
9	3.0
11	1.0
12	0.5
13	0.0
15	0.0
16	0.0

Cell population density in the central portion of tubo-promontorial pad (TP) as a function of age.

tion density is thus due in part to resulting increased intercellular distance, but no doubt cell destruction plays a significant role here also. The tissue appears increasingly "diffuse" and acellular (see Fig. 11) with increasing age until, finally, holes begin to appear and it assumes an increasingly web-like appearance (see Fig. 5). It would seem as though the mesenchyme were under the influence of some stimulus to fill the expanding cavity without being able to proliferate cells at a rate rapid enough to maintain its original cell concentration.

Table IV shows corresponding data for the tubo-promontorial mesenchyme pad.

Comparison of Tables III and IV reveals a slightly lower initial cell density in the TP region as compared with the S region. In the TP region there is a quite marked drop on

* This anomaly may well be the result of marked individual variation, not cancelled out because, in the series studied, each age was represented, on the average, by less than two specimens apiece.

the third day (*cf.* fifth day in the S region). The differences in cell population of the two regions thus parallel the earlier overall volume changes in the TP regions as compared with the S region.

During the above study, differentiation changes were noted in the mesenchyme cells with increasing age. Most obvious is the laying down of parallel rows of spindle-shaped cells in the deepest layer of the promontorial pad (see Fig. 12).

More subtle is the appearance with increasing frequency of two types of cell within the primitive syncytial reticulum itself. One type has histiocytic* characteristics (see Fig. 13), and it may well be that such cells individually survive the disintegration of their early home by wandering off (*cf.* L. K. Guggenheim's[5] description of "reversible tympanal fibrosis" in children).

The other cell type shows progressively increasing vacuolization. Early, this consists only of a barely visible stippling of the cytoplasm, progressing to a perinuclear rosette of small vacuoles, and finally to the formation of typical signet ring cells resembling fat cells (see Figs. 14 to 18).

A quantitative study of differentiation of mesenchymal cells

* Since the histiocyte is still a pluripotenital cell, its derivation from a stellate mesenchymal cell probably represents a "modulation" in the sense of Paul Weiss, rather than a true differentiation—which implies an irreversible reduction in repertoire.

Fig. 11. Rat at nine days. Promontorial mesenchyme pad showing diffuse, homogeneous appearance of ground substance and sparsity of cells prior to disintegration. (Oil immersion.)

Fig. 12. Rat at eleven days. Developing periosteum beneath the promontorial mesenchyme pad. Note typical stellate mesenchymal cells above this layer. (Oil immersion.)

Fig. 13. Rat at nine days. Nest of histiocytic cells in disintegrating promontorial mesenchyme pad. (Oil immersion.)

Fig. 14. Rat at two days. Stellate mesenchymal cells from promontorial pad, showing early vacuole formation. In some instances, a perinuclear arrangement is already evident.

Fig. 15. Rat at 1 day. Mesenchyme cells showing peri-nuclear arrangement of slightly larger coalescent vacuoles.

Fig. 16. Rat at 12 days. Mesenchyme cell showing much larger coalescent vacuoles. Peri-nuclear arrangement not exclusively exhibited here, cell being a blown-up version of a stellate form, rather than rounded.

Fig. 17. Rat at one day. Rounded mesenchymal cell with very large coalescent vacuoles. Note nucleus barely visible. This form approaches the typical signet ring cell.

Fig. 18. Rat at 12 days. Signet ring cells.

Fig. 11.

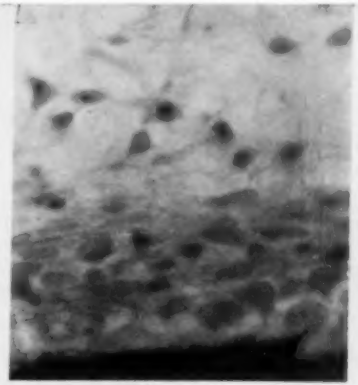

Fig. 12.

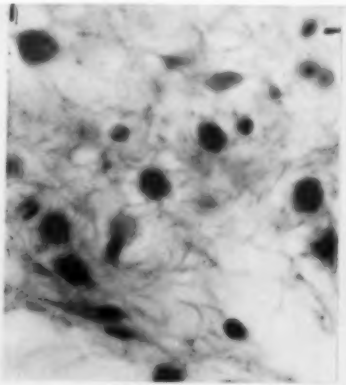

Fig. 13.

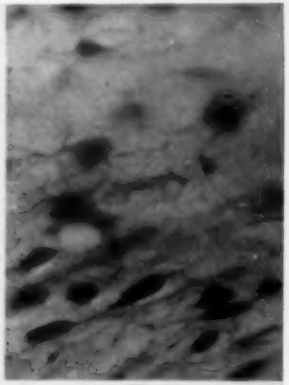

Fig. 14.

Fig. 15.

Fig. 16.

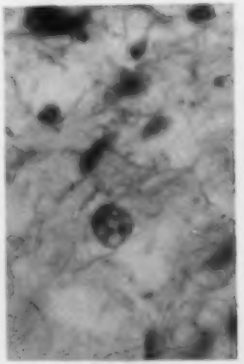

Fig. 17.

Fig. 18.

into histiocytes and vacuolated cells was carried out for both TP and S regions. It was noted that these "differentiated" cells existed in unevenly distributed clusters. Hence selection of areas for cell counts involved a highly subjective choice factor. For this reason, a small area was chosen in the S region which showed a great stability in size and configuration throughout the series of animals studied: an area bounded by the stapedial artery, footplate and anterior crus. The increase in the two types of so-called "differentiated" cells mentioned above as a function of age is shown in Table V.

TABLE V.

Age of Animal in Days	Macrophages	Vacuolated Cells
1	0	0
2	0	0
3	0	0
5	0	0
7	1	1
9	5	2
11	7	7
12	7	8
13	11	6
15	11	8
17	10	9
18	20	10

Numbers of differentiated cells as a function of age. Area bounded by anterior stapedial crus, stapedial artery and footplate.

The increase in the number of differentiated cells as the age of the animals increases is quite apparent. A comparison with Table III, which lists cell densities for this region, indicates that the increase of differentiated cells occurs at the same time that the total number of cells is decreasing. In the older stages noted in Table V, practically all the cells show some degree of differentiation.

The corresponding study in the TP region is complicated by the fact that the mesenchyme here is considerably less homogeneous. Three layers are apparent from the first day of life and become increasingly distinct with advancing development:

1. The deepest layer, next to the perichondrium* (later, periosteum) of the promontory;

2. The central layer, which constitutes the bulk of the total mass; and

3. The layer adjacent to the free luminal border of the mass.

Layers 1 and 3 appear not only to have a more condensed intercellular substance than Layer 2, but a higher total cell population and a higher incidence of differentiated cells. These data are shown in Table VI.

TABLE VI.

Age Animal In Days	Layer 1 (a)	(b)	(c)	Layer 2 (a)	(b)	(c)	Layer 3 (a)	(b)	(c)
1	25	4	0	11	1	1	21	0	0
2	11	0	0	7	1	0	18	2	0
3	20	0	0	8	1	0	22	7	0
5	9	0	0	3	0	0	20	6	0
7	10	0	1	3	1	0	12	3	0
9	10	4	0	5	0	0	7	3	0
11	5	1	0	1	0	1	4	2	1
12	32	3	6	3	0	0	42	12	8
13	0	0	0	0	0	0	0	0	0

Changes in three layers of the promontorial mesenchyme pad:
Layer 1 is the layer next to the perichondrium-periosteum of the cochlear promontory.
Layer 2 is the large, inert central portion.
Layer 3 is the layer next to the free tympanic lumen.
(a) Total cell population.
(b) Histiocytic cell population.
(c) Vacuolated cell population.

It would appear that the large and apparently inert middle layer of the promontorial mesenchyme pad distintegrates with the least exhibition of "differentiated cells." Consequently the formation of histiocytes and signet ring cells can hardly be the principal mechanism of disposal of this layer. It may well be that an enzymatic liquefaction of the stroma occurs, presumably involving a depolymerization of the ground substance.

Converse changes in the ground substance of the *actively*

* A very thin perichondrium is easily distinguishable on the first day of life. At 11 days (see Fig. 12), this layer is notably thickened. Undoubtedly mesenchymal cells are here becoming periosteal fibroblasts. For purposes of this quantitative study, the perichondrial-periosteal layer is excluded from the mesenchyme mass as now constituting a definite structure separate from it.

growing mesenchyme of placental villae were studied by Gersh and Catchpole.[18] Of special interest was the finding of intracellular glycoprotein granules which reached a maximum population density at four months, corresponding to the time of maximum blood and urinary content of chorionic gonadotropin. The fetal placental mesenchyme cell was thought perhaps to be a specific target of this hormone. The number of granules declined from four months to term, when only rare cells exhibited them.

The significance of the vacuolated and signet ring cells was not at first apparent since fat, as such, is not normally found in the tympanic cavity. (Dorothy Wolff[2] described fat in the sinus tympani of two autopsied children, ages 13 months and eight years). The appearance of fat droplets in mesenchyme cells was apparently first described by Lewis[19] in 1918. She explanted 6 to 9 day chick embryos into nutrient medium containing Sudan III, and observed that "the fat droplets of the mesenchyme cells remain distinctly smaller than those of the clasmocyte." (Her purpose appears to have been to show that these droplets did *not* arise as a result of mitochondrial differentiation.) It goes without saying that Sudanophilia is not today considered conclusive evidence of the lipoid nature of an intracellular inclusion. Even the signet ring cells observed by us, which were first described in mesenchyme as early as 1925 by Ernst,[20] cannot simply be assumed to be fat cells. Further histochemical investigation is now underway in an effort to settle this question. Pending results of this study, we have received considerable enlightenment from the work of Holtfreter[21,22,23] on the transformation of lipochondria into the larger liposomes by enzymatic proteolysis of their surface films, a process to which he has given the name "lipophaneresis." The appearance of larger cytoplasmic inclusions of unmasked fat due to coalescence of smaller inclusions was described by Bungenberg De Jong,[24] who called it "coacervation." Holtfreter[22] states that the appearance of fat droplets, vacuoles and granules is common in physically irritated cells, *e.g.*, in ageing tissue cultures. Their presence is pathological, but does not necessarily lead directly to the death of the cell. The unmasking of lipids is apparently due to liberation of an enzyme normally inactive because combined with other cyto-

plasmic compounds. These considerations lend weight to the hypothesis that mesenchyme dissolution in the middle ear is an enzymatically determined process, and a mechanism is suggested by means of which the dilatation of the tubal orifices with the Gyergyai olives might be effective: liberation, by compression, of a proteolytic enzyme either from the epithelium of the dilated region or from submucosal remnants of unresorbed mesenchyme beneath it. Further work, it is hoped, will put this hypothesis to a test.

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ODONTOPATHIC ASPECTS IN OTOLARYNGOLOGY*†

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Many patients present themselves to the otolaryngologist with problems that have as their source dental structures. It is these problems that will be discussed, especially in regard to diagnosis and complications.

PAIN OF DENTAL ORIGIN.

Facial Pain — Facial pain often presents a perplexing problem, not only to the patient, but also to the otolaryngologist. He should be able to evaluate dental structures to some degree and should be aware that the teeth may be the etiology in pain about the face.

Inflammation of the internal soft tissues of the teeth, known as pulpitis, may be the source of vague facial pain. The patient sometimes is unable to localize an offending tooth. Usually pain referred to the ear comes from a mandibular tooth; pain in the eye or upper part of the face has as its source, an upper tooth. This is not an absolute rule, and in an occasional instance pain may differ entirely from this pattern. The patient may complain of what he calls "headache" about the temporal and facial regions. Since the dental pulp responds to irritation by inflammation, it is necessary in examination to note, *1.* whether caries are present; *2.* whether recent dental treatment has been received. Dental restoration may have resulted in inadvertant injury to the pulp through its exposure, excessive heat, or irritation by certain fillings, such as the silicates. Traumatic occlusion by a high spot on

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† Presented as a projected clinic before the Chicago Laryngology and Otology Society, April, 1956.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication, June 25, 1956.

the filling may cause irritation. The latter group, especially, may be puzzling to the otolaryngologist, since he may see a well restored tooth and not suspect that this tooth with its pulpitis is the source of pain.

The use of thermal stimuli is a great help in diagnosis. The normal pulp pain response to extremes of heat and cold leaves in a few seconds, as soon as the stimulus is removed. In an inflamed pulp the response persists. For measuring fine differences in threshold of response an electric pulp tester is needed.

As the inflammation persists, necrosis takes place, and the patient may be comfortable for varying periods of time (days to months, even years) until the necrotic tissue of the pulp becomes infected with enough virulent organisms to involve the tissues surrounding the tooth, resulting in periapical infection, abscess, osteitis or osteomyelitis. The affected tooth responds to percussion by increased tenderness as compared to percussion of the other teeth. Percussion is an easy and accurate means of localization of periapical involvement. The otolaryngologist can easily use the handle of his laryngeal mirror to tap several teeth in evaluating gross differences. Since the nerve tissue of the pulp has died, there is little reaction of the tooth at this stage to thermal changes. The next step is spread of the infection as a cellulitis or formation of an abscess. It might be well to point out that after a periapical abscess has drained into the facial soft tissue from within the bone, percussion tenderness may be absent; but the facial swelling is often characteristic. The parietal abscess that is located lateral to the tooth and is often secondary to periodontal disease may be readily localized by the inflamed surrounding gingiva. It is frequently associated with at least some sensitivity to percussion, although the tenderness to percussion may not be outstanding.

Post Extraction Osteitis — Recent extraction of teeth may present a problem of diagnosis. A patient comes to the otolaryngologist with the history that he has a one-sided facial pain for which a tooth had been extracted with temporary relief; and now having a return of dull aching facial pain,

four to five days after the extraction, he seeks another cause. This is the typical story of the "dry socket" or post-extraction osteitis which usually subsides spontaneously within about 14 days. The patient may be given relief and at the same time a therapeutic test, by cleansing of the tooth socket and the use of a paste ointment containing an antiseptic and topical anesthetic. The presence of fever and continual pain may mean extension of infection, and osteomyelitis must be considered.

Unilateral Pharyngitis — The discomfort of a unilateral sore throat or pharyngitis, especially in a young adult, may have as its etiology a pericoronitis or inflammation about a partially erupted third molar. This is usually caused by infection under the remaining flap of tissue, and frequently is associated with a certain amount of trismus. The diagnosis of this condition and its complications (which will be discussed later) is of much importance to the otolaryngologist. Occasionally unilateral pharyngitis may be secondary to dental treatment. Inflammation or hemorrhage within the internal pterygoid muscle may occur as the result of the use of local anesthesia in a mandibular block. Fortunately, infection, broken needles or injection of wrong solutions are now relatively rare complications, even though the use of local anesthesia has increased in restorative dentistry.

Impacted Teeth — An impacted third molar may cause vague and variable discomfort. Impacted teeth in themselves, however, may be completely symptomless. These teeth are often malformed and malpositioned, hidden completely or partially by bone and soft tissue. Thus, they may cause discomfort by pressure on the mandibular or maxillary nerves or on adjacent teeth, even rarely causing exposure of the pulp of the adjacent tooth. Adequate X-ray studies of the third molar area are necessary to note the presence and status of an impacted molar. Other teeth, especially the upper canines, may remain unerupted and hidden to cause facial pain.

Temporomandibular Joint — Pain about the face and ear may be associated with the temporomandibular joint. It is important to remember that this area may be affected with the same diseases and conditions that affect the other joints

of the body. The jaw muscles, ligaments and articulating surfaces may be subjected to traumatic, inflammatory and degenerative diseases.

The history, especially for evidence of degenerative and systemic arthritis, and recent or old trauma is important. Knowledge of a recent difficult mandibular dental extraction, surgery in the oropharyngeal regions, especially under general anesthesia, and previous facial fractures involving the mandible are important.

Examination of the joint for subluxation should be tested. Pain may be produced when a finger is placed over the joint as the patient opens his mouth and also moves the mandible laterally. This helps in diagnosis. Frequently the muscles and ligaments are the source of the discomfort rather than the articulating surface. Selective injection of a local anesthetic in various sites, as in the joint and/or its associated muscles will help in localization. X-ray examination should be carried out in the diagnostic workup of temporomandibular joint pathology. Frequently this will be disappointing.

Evaluation of dental occlusion is important, mainly because imbalance of occlusal function leads to traumatic strain of the ligaments, muscles and articulating surfaces of the temporomandibular joint.

Treatment is based on diagnosis and removal of underlying causes. This may include treatment of a psychic tension state in bruxism, establishment of balance occlusion where indicated and/or systemic medication in the infections and degenerative arthritis.

As with any joint disease, joint rest is essential. The patient should be discouraged from experiments with jaw movements to reproduce the pain. Chewing gum, pipe smoking and similar habits should not be permitted. Often the only method of obtaining joint rest is by inter-maxillary wiring. In acute states, injection of a cubic centimeter or more of Hydrocortisone with procaine into the joint is useful. Application of heat aids in decreasing muscle spasm. Salicylates still remain valuable therapeutic agents. The otolaryngologist should be

aware that surgical procedures, such as meniscectomy and condylectomy, may become necessary. Rongetti suggests that a rational approach for meniscectomy is through the endaural route. A sclerosing agent injected into the temporomandibular joint in excessive subluxation has been used with some degree of success.

Stomatitis — Stomatitis resulting from various infections and oral manifestations of systemic disease is not within the province of this paper. The otolaryngologist should be aware, however, of the irritations and allergic idiosyncrasies that may be produced by medications, mouth washes, dental plates, dental powders and dentures. Materials containing synthetic detergents and denture plastics that are not completely polymerized (resilient plastics) are the most frequent offenders.

SURGICAL CONSIDERATIONS.

Space Abscess — Let us consider now the space abscess and what it represents clinically. It may be defined for our purposes as a collection of pus within the connective tissues of the face. Numerous types occur, but our discussion is confined to those related to the back part of the jaws. These are the pterygomandibular, infratemporal, parapharyngeal and parotid space abscesses.

An otolaryngologist is familiar with the toxic adult patient who is unable to open his mouth. His first thought is a peritonsillar abscess or peritonsillitis, for these often limit opening. Within a few centimeters of these structures, however, lie the third molars, or wisdom teeth. Infection arising from these may also restrict movement of the mandible and cause severe pain. As stated above, inflammation surrounding the crown of the third molar occurs. This inflammation or pericoronitis, may extend some distance from its source. Its spread into inaccessible areas, where abscess can form, becomes a major complication of pericoronitis.

It should be explained before continuing, that the description of so-called spaces is for simplicity. They are not spaces at all, but only potential spaces. These connective tissue compartments intercommunicate, one blending into the other.

Pterygomandibular space abscess is the first to be discussed. It is found between the mandible and inferior part of the internal pterygoid muscle. A patient may suffer for days, because the doctor cannot open the patient's mouth to see what is happening. A preceding pericoronitis is usually the cause, but sometimes needle borne infection is carried into the space. Aspiration is helpful in establishing a diagnosis before incision is performed. Treatment may be attempted under local anesthesia, but occasionally general anesthesia may be needed to gain access to the mouth.

Infratemporal abscess is the second complication of pericoronal infection. This area lies between the vertical ramus of the mandible and the upper portion of the pterygoid muscle. It is necessary, then, to differentiate carefully since each abscess is incised in a different place. This infection, limited by bone and muscle tissue, does not produce extensive swelling unless the infection spreads toward the temporal fossa externally, or toward the pharynx internally. A thrombophlebitic process of the pterygoid and pharyngeal plexuses must be considered when swelling in the oropharynx occurs. Incision and drainage, usually under local anesthesia, is the definitive treatment when this is possible. In extensive infections a combined internal and external approach may have to be employed.

Parapharyngeal space abscess is a third complication of pericoronitis. The anterior compartment of this space is located between the internal pterygoid muscle and the superior constrictor of the pharynx. A posterior part is also present, but this is not as often involved. As with the others, trismus is again a prominent sign. Severe pain may occur with swallowing, and peritonsillar abscess must be differentiated. Antibiotics are administered liberally as medical treatment is tried, and by the time the patient is seen by the otolaryngologist, the picture has been complicated. Incision and drainage are indicated, but in this area spontaneous drainage or resolution will at times occur.

A fourth infection is simply that between the two connective tissue layers surrounding the parotid gland. Severe pain in the

parotid area develops. Differentiation from a parotid tumor is important and not always easily determined, especially in this antibiotic era.

Submaxillary space infection usually occurs as extension from apical abscess of bicuspid and first and second molars. It may also occur, however, from pericoronal infection in third molars.

Abscess in the spaces discussed include the more difficult types seen by the otolaryngologist. They may be summarized as follows:

1. Space abscess in the tissues of the face may occur concurrently or weeks following pericoronal or apical infection of third molar teeth.

2. Trismus is important in diagnosing space abscess, and odontopathic factors must be considered.

3. Abscess in regions limited by bone and muscle cannot produce typical swelling, and so diagnosis may be difficult.

4. Prudent surgical and supportive measures based upon understanding of the anatomy is preferable.

Oro-antral Fistula — A few clinical considerations of oro-antral fistula may now be mentioned. Many small fistula close spontaneously after extraction. The large rooted first permanent molar and the double rooted first bicuspid are favorites in fistula formation. Removal of upper third molars may also cause a fistula. A tiny opening into the sinus in this region is easily missed. This is seen only as a dimpling which simulates healed and scarred mucosa. The course of the tract may be tortuous and seen only with radio-opaque injection. Bizarre symptoms of sinusitis may occur.

Fistula may be associated with loss of a tooth or root into the sinus. Sublabial antrotomy may become necessary to retrieve it. As large as a tooth or root is, it may be hidden beneath the sinus mucosa and found only after frustrating search. Before exploration the lateral as well as the antero-posterior X-ray should be examined. The tooth may have been

forced into the pterygomaxillary fossa instead of into the sinus.

Treatment of an oro-antral fistula may be accomplished by a pedicle flap from the buccal or palatal mucosa. Closure is dependent upon good drainage and absence of pathology in the maxillary sinus, and a nasal antrotomy may be performed where indicated. Simple approximation after freshening the edges of a fistula in a delayed closure is usually doomed to failure.

Facial Trauma — Facial trauma is a big subject. Only a very few odontologic matters of interest to the otolaryngologist can be mentioned here. Early tracheotomy is a preliminary consideration in extensive facial damage. In jaw fractures the simple examination of the mouth reveals much and may tell as much as the battery of X-rays usually ordered. Early treatment of certain jaw fractures, the same day if possible, is advantageous. Treatment may necessarily be delayed for days before the patient is able to tolerate therapy. Neurologic complications usually take precedence over definitive therapy. It is not necessary, however, to neglect the patient suspected of intracranial damage. Co-operation is needed in maintaining an adequate airway and in preventing aspiration of teeth or other foreign bodies. Sometimes temporary immobilization of fragmented bones is necessary. Middle face fractures may be reduced early. A shock state is overcome, and bleeding from the face often subsides when the bones are reduced.

Function of the jaws is based upon occlusion of the teeth, or proper interdigitation. It is to the advantage of the otolaryngologist to appreciate the importance of occlusion.

Retention of a tooth in the line of fracture is desirable when it may reasonably be saved. Conversion of an otherwise simple reduction into a more complex one can occur by the untimely removal of a tooth. In mandibular fractures, for instance, the only deterrent to extensive displacement of the vertical ramus of the mandible may be a lone molar at or near the line of fracture.

Blood in the ear in condylar fracture is not uncommon. Cranial damage must be suspected and due care taken. On the other hand, the blood may be from a tearing and fracture of the antero-inferior tissues of the ear canal by the condylar head as it is driven against the external canal. The otolaryngologist has a definite advantage in evaluation. Correctly used, the knowledge leads to a more prompt reduction of the jaw and anterior ear canal wall. Attention to the complication may avoid a later stenosis of the ear canal.

Many approaches to the treatment of jaw fractures have been proposed. Immobilization is essential. Intermaxillary wiring is useful and simple. Elastic traction, using small rubber bands between the upper and lower arches is an excellent way to reduce fractures.

Both open and closed reductions of the mandibular condyles and other parts of the jaws should be used as indicated. In extensive fractures other devices, sometimes quite complicated, may have to be used. No one way to reduce fractures is best, but many are equally good.

Aspirated Foreign Bodies — Dental foreign bodies may occasionally be aspirated into the air or food passages. Holinger in one series reported 2.5 per cent of foreign bodies in the respiratory system were dental objects. This is a surprisingly low figure, considering how often the oral cavity is invaded for dental treatment.

Aspirated teeth, or parts of teeth, account for the largest number of dental foreign bodies. Extraction under general anesthesia is a greater risk than extraction with local anesthesia. Roentgenographic evidence is helpful in locating dental objects. Not all foreign bodies are radio-opaque. Aspirated dental calculus, for instance, may be laden with infective material, and will hardly show up in a chest film. Silicate cements, used in the restoration of dental caries in anterior teeth are easily penetrated by Roentgen rays. Small pieces of plastic appliances, known as acrylic, will not produce a significant shadow. Metals and teeth will ordinarily be seen on neck and chest films, though they may be hidden by heart, liver or bone.

Osteomyelitis — Osteomyelitis of the jaws is difficult to treat, and the otolaryngologist will be called upon for help and counsel by his medical and dental colleagues. The cause is usually of odontogenic or traumatic origin, and the mandible is the most common site affected. In infants the maxilla is more commonly affected. During the acute stage much reliance is placed on antibiotics. Emphasis should be given to the surgical aspects after the acute phase has subsided. Sequestrectomy and saucerization are useful and may be curative in the treatment of osteomyelitis of the jaws. With the safeguards of pre- and postoperative antibiotics, the source of infection in bone can be more readily controlled.

Radiation to the jaws may produce an osteomyelitis. Carious, and sometimes sound teeth, may have to be removed before the jaw is irradiated in order to avoid difficulty later on. Irradiated bone does not heal well, and very resistant and painful osteomyelitis results when teeth become infected and have to be removed.

ODONTOGENIC TUMORS.

We may now deal briefly with swelling due to cysts and benign tumors of odontogenic origin. Malignant tumors and those arising from non-odontogenic origin are not included.

Odontogenic Cysts — Odontogenic ectodermal cysts may arise from the follicle of the tooth or from the periodontal membrane. Several types of follicular cysts occur. The first is the primordial cyst, which forms from the degeneration of the stellate reticulum in the enamel organ before any calcification takes place. Another type is the dentigerous cyst. This originates because of degenerative changes in the reduced enamel epithelium of the developing tooth. It is a closed epithelial lined sac formed about the crown of an unerupted tooth. It is most commonly associated with impacted teeth, such as the third molar or canines. Multilocular cyst is a third kind of ectodermal odontogenic cyst. This forms from adjacent tooth germs or proliferation of epithelium.

The other main group of cysts are those arising from the periodontal membrane and are named according to their

location. Those at the apex of the tooth are termed periapical, while those located laterally are called lateral peridontal cysts. Their epithelium is derived from remnants of the sheath of Hertwig. Frequently these cysts are preceded by chronic proliferative inflammation. If acute infection is present, then all the symptoms of an acute abscess are present in the clinical picture. Cysts are important because of their slowly expanding nature. Much bone destruction and loss of function of the jaws may occur unless they are recognized and treated adequately.

Benign Tumors — As with cysts, benign tumors may also arise from developmental dental structures or from closely related peri-dental tissue. Classification may be complex. Only the more common types will be mentioned.

Inflammatory hyperplasia may often be confused with tumors. These are sometimes termed epulis. There is a tendency to recur if the tissue and underlying cause is not removed. Hyperplasia of the gingiva occurs because of irritation of a flange of a denture, irritation by an overhanging filling, decayed teeth, or calculus beneath the gingiva. Hyperplasia of a non-inflammatory nature may also occur in response to stimulation by certain drugs. This is most commonly seen in dilantin therapy for epilepsy.

Fibromas develop in the gingival or interdental papilla. They also form from periosteum or periodontal tissue, and may be pendunculated or sessile. A fibroma may be seen at the maxillary tuberosity, and may be difficult to distinguish from malignancy when trauma has produced ulceration. Some fibromas show osteogenic activity, and may be extremely firm.

Another benign tumor is the peripheral giant cell tumor, also termed an epulis. The classical type has a bluish red tinge, due to vascularity and deposit of hemosiderin in the tissue. It frequently forms between the teeth, causing a separation and sometimes invasion of the underlying bone. It occurs as both a sessile and pedunculated tumor. Rare cases have occurred where fibrosarcoma has developed. In pregnancy similar tumors are found. It is believed these latter

occur because of hormonal stimulation during pregnancy. They usually recede after parturition.

Among the most common benign osteogenic tumors are the torus palatinus and mandibularis. The palatal tumor is in the midline, and easily recognized. The mandibular tumors occur on the lingual surfaces of the mandible near the bicuspid teeth. They are usually smooth, very hard, and have been present for a long time. Often the patient is unaware of the mass in his mouth, until it has been called to his attention by his dentist or physician. These are considered to be hypertrophic bone or osteomas, and do not have to be removed unless they interfere with placement of a denture or other dental appliance.

SUMMARY.

Pain of dental origin, with some surgical considerations of interest to the otolaryngologist are discussed. Certain odontogenic tumors are considered.

It may be emphasized that the discussion presented is merely an introduction to that portion of medical science which the otolaryngologist finds necessary to evaluate and correlate with his own problems.

1648 East Ave., Berwyn, Ill.

UNIVERSITY OF ILLINOIS, COLLEGE OF MEDICINE.

The next Laryngology and Bronchoesophagology Course to be given by the University of Illinois, College of Medicine, is scheduled for the period November 5 through November 17, 1956. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois, College of Medicine, 1853 W. Polk Street, Chicago 12, Ill.

STATISTICAL ANALYSIS OF THE STAPES MOBILIZATION PROCEDURE*.

CHARLES E. KINNEY, M.D.,

Cleveland, Ohio.

When I started to put down on paper a comparison of the stapes mobilization procedure with the fenestration operation it was done in order that I might honestly and intelligently inform my patients as to what they might expect. It soon became apparent that I would have to call on some of my colleagues for help, because my experience was quite limited. A questionnaire on this procedure was sent to 14 men whom I either knew were doing the procedure or whom I thought might be doing it. At the outset, let me make mention why I did not send a questionnaire to Dr. Sam Rosen. Everyone knows that he was the one who revived this procedure and for the first time in otological surgery really made it work. His techniques and results have been very adequately publicized. He was very kind to me when I visited him year before last, and I publicly thank him for stimulating my interest in this operation; however, I wanted to find out what other persons could do with the procedure.

One of the remarkable things about this survey is that all of the 14 questionnaires were returned. Three men said that they had not done the procedure as yet; therefore, the following analysis represents the combined work of these men:

Richard J. Bellucci, M.D.; Edward H. Campbell, M.D.; Eugene L. Derlacki, M.D.; E. P. Fowler, Jr., M.D.; Victor Goodhill, M.D.; Frederick Harbert, M.D.; Robert Henner, M.D.; Howard P. House, M.D.; Charles E. Kinney, M.D.; Clair M. Kos, M.D.; George Shambaugh, M.D.; Theodore E. Walsh, M.D.

To these men, I give my thanks for their cooperation in this endeavor.

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* Read before the University of Florida, Post Graduate Seminar in Otolaryngology, Miami Beach, Fla., Jan. 21, 1956.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication, February 15, 1956.

The first question had to do with the number of cases which each man had done. The total reported cases was 1117. The second question was "Irrespective of the type of case, what is the percentage of the whole total in which there was a successful *audiometric* improvement of hearing within the first three to four weeks?" No attempt was made to set up a uniform standard of "successful improvement". They were asked to use their own criteria, because each one of these men had been doing the fenestration operation for a relatively long time. The third question was the same as the second question, except that the post-operative time was over six months. In analyzing the answers to these two questions, it was felt that it would be more accurate to balance each man's results against the number of cases that he reported; therefore, his percentage answer was multiplied by the number of his cases. These two figures were then totaled and the total number of cases was divided into the total percentages. The final figures were:

Successful three to four weeks—53 per cent.

Successful more than six months—40 per cent.

When one tries to obtain further ideas from these figures, particularly the added comments to these two questions by some of the men, there are two points that stand out: first, is that although the time consumed in doing this procedure is considerably shorter than the average time for fenestration operation the meticulousness required in order to obtain better results is of far greater importance than with the fenestration. The second point is, that as one does more and more cases his percentage of successful results goes up. For instance, in House's series of cases he reports that in his first 25 per cent of cases he only had 35 per cent successful results; in his second 25 per cent of cases his successes were 50 per cent, and in his last 50 per cent of cases his successes were up to 60 per cent. The observations alone have been sufficient to encourage me to continue this work and try to improve upon my earlier results.

The next question that I wanted some help on had to do with the idea that was created in the minds of the public by statements in newspaper and magazine articles to the effect

that if the mobilization procedure did not work one could always do the fenestration operation on that ear.

It has always been my contention that the second most important factor in obtaining a really good result from a fenestration procedure was the maintenance of a completely intact and normally functioning ear drum. Of course the number one factor is the creation of the fenestra itself; therefore, I wondered whether the elevation of half of the drum by a mobilization procedure would leave a normal functioning drum after the incus has been removed and the head of the malleus clipped, as is done in the fenestration procedure.

In this series of 1117 cases, there were 60 per cent unsuccessful results after six months, or 670 cases that were potential cases for a fenestration. There were reported 40 cases that had a fenestration done on a previous unsuccessfully mobilized ear. This is only 5.9 per cent of the potential cases; however of these 40 cases 33 were successful, which is an average of 82 per cent, quite an admirable figure. When one tries to draw some conclusions concerning this question he runs into several unknowns. Of the 40 fenestrated cases (5.9 per cent) the results were very good; but what about the 630 other potential cases (94.1 per cent)? It is safe to assume that all of them wanted better hearing or they would not have subjected themselves to the original mobilization procedure. Some of them have not had sufficient time interval after the mobilization. Some of them have not had a fenestration because of economic reasons; however, all the men felt that, except in the very rare case, the condition of the ear drum after a mobilization attempt would not be a contraindication to a future fenestration. The 82 per cent of successful fenestrations in those cases that had been previously operated would tend to support this view.

The next question had to do with the amount of the bony posterior canal wall that had to be removed in order to have adequate vision of the stapes. Here again it was felt that each surgeon's answers should be balanced against the number of cases he had done. The results of this calculation reveal the following:

15 per cent required no bone to be removed from the posterior wall;

79 per cent required 1 to 2 millimeters to be removed;

6 per cent required more than 2 millimeters to be removed.

The next question had to do with the necessity of operating table audiometry. Nine of the 12 men whose cases are reported in this series felt that some sort of operating table hearing testing was necessary. Five of these nine felt that it was *absolutely necessary* to use some method of electrical audiometry at the time of the operation rather than depend on his own voice testing. It is interesting to note here that these five men reported higher percentage of good results than the other seven men.

Goodhill uses a standard commercial pure tone audiometer to do his operating table threshold testing. I think, but am not completely sure, that he uses only the frequencies of 500 and 1000. He simply puts a sterile towel over the auricle each time he tests the patient's response.

House has devised his own "Otometer," which is a pure tone instrument more adaptable to use in the operating room than a commercial audiometer. House feels that pure tone testing on the operating table is more reliable than electrically controlled word testing. He also feels that the amount of operating room improvement does not indicate the amount of improvement in hearing that the patient may have a month later. This idea is not shared by the other men.

Both Goodhill and House point out that in addition to an office pre-operative threshold for this particular ear, a premobilization threshold must be established after the lower posterior part of the ear drum has been elevated and then returned to its normal position. This must be done each time one tests the hearing. The other three men who said operating table electrical audiometry was an absolute essential did not delineate the exact kind of testing that they employ; therefore, it is apparent that one must do the stapes mobilization under local anesthesia, and that one's vision and sense of

touch are not sufficient to evaluate the degree of mobilization that has been accomplished.

My next question had to do with the most effective point to apply one's force in order to mobilize the footplate of the stapes. It is interesting to note that all 12 men seemed to be in agreement that fracture of either one or both of the stapedial crurae before the footplate is at least partially mobilized is still the biggest stumbling block to the success of this operation. Five of the 12 men reported that they used their first mobilizing force on the head of the stapes. Two men exert their initial force on the incus.

Goodhill advocates using a type of cement between the incus and the stapedial head if this joint should be dislocated in the original probing before one's actual mobilizing force is exerted. The other five men use their original force on the neck of the stapes as originally advocated by Rosen. There was absolutely no agreement as to the direction in which this force should be initially applied, that is, whether it should be a push or a pull; however, there was complete agreement that it must be in the axis of the two crura and that the exact direction of this axis must be ascertained before the original force is applied. This is a dictum that was first pointed out by Rosen. What one does after there is either visual or palpable evidence that there has been a fracture of one or both crurae, and before a hearing test shows that there has been some mobilization of the footplate, remains a big question.

Kos and House report that they use a small pneumatic hammer to produce a series of blows to parts of the rim of the footplate in case there has been a fracture of a crura. Three of the other men in this series report that they are working with this technique. It is obvious that this is the ultimate in technical surgical perfection, although House reports that by following this method he is able to mobilize at least part of the footplate in 95 per cent of his cases at the present time. There is one positive conclusion that can be drawn from consideration of this item: namely, this operation is *not the ten minute office procedure,* which idea has been bantered about too much in both medical and public discussion. There was rather universal agreement that no one should attempt this

operation if they were not already fairly adept at doing the fenestration.

My next questions were directed towards the matter of complications. There were reported 19 cases of temporary acute otitis media. This is somewhat under 2 per cent of the total, and is certainly a tribute to the technique employed. There were no reported cases of semi-permanent or chronic otitis media and no cases of facial paresis or facial paralysis. There were three cases of progressive perception loss of hearing after this procedure which was assumed to have been caused by a hemorrhage into the perilymph. This is an insignificant number, but it can happen.

At this point in my questionnaire, I interposed a purely philosophical question: "Do you think that this operation will subsequently replace the fenestration operation?" Twelve answered "no" to this question, most of them emphasized in one manner or other; however, five of the 12 men said that they recommended that mobilization be tried first in all cases suitable for fenestration. There was rather complete agreement that mobilization had priority over fenestration in certain instances, such as economic reasons, cases that had a fair amount of perception loss but still a fairly good cochlear reserve, elderly patients or those with other constitutional diseases, and in cases that have had a fenestration in the other ear.

SUMMARY.

1. The complete cooperation and frankness of the surgeons that answered this questionnaire is something unique in medical annals.

2. 1117 cases of stapes mobilization are reported.

3. The immediate postoperative successful results were 53 per cent and the long range successful results were 40 per cent.

4. There is good reason to believe that some additional refinements in the technique of the actual mobilization will be developed which will certainly increase the chances of any one person obtaining a successful result.

5. Operating table hearing testing is a must, and this should be done audiometrically.

6. While the technique required to do this operation is more delicate than that required to do a fenestration, the time consumed is shorter, and certainly the post-operative care of the patient is reduced considerably.

10515 Carnegie Avenue.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

Washington, D. C., May 5-10, 1957.

The Officers and the Organizing Committee of the Sixth International Congress of Otolaryngology cordially invite you to attend the Congress which will be held in Washington, D. C., U. S. A., May 5 through 10, 1957.

REGISTRATION AND OPENING.

Any qualified physician may become a Member of the Congress by paying a registration fee of $25.00, U. S. A. Non-medical personnel may register as Associate Members for a fee of $10.00, U. S. A. The registration fee includes the privilege of attending all official meetings of the Congress except the banquet for which an additional charge will be made. Those planning to attend the Congress should complete *Form A* and return it to the General Secretary.

IDENTIFICATION OF HEARING LOSS BY THE CLASSROOM TEACHER.

FRANK KODMAN, JR., Ph.D.,

Lexington, Ky.

The purpose of this investigation is to compare audiometric findings with teacher identifications of hearing loss to determine the accuracy of the latter method. Estimates of the incidence of mild-to-severe hearing losses in elementary school children based on studies by Curry,[1] Goldman[2] and Newhart[3] would be on the order of 10 to 25 per cent. Arbitrarily, mild losses may constitute audiometric thresholds 15 to 25 db at one or more frequencies in either ear. One would expect most of these losses to be conductive and worthy of medical attention from the standpoint of health and hearing conservation.

The teachers were asked to identify hearing losses 15 db or greater for the reasons cited above. Admittedly, the identification situation is unfair to both the teachers and the pupils; nevertheless, the classroom teacher is often expected to detect pupils whose auditory sensitivity is suspect. In a similar study, Curry[1] found that approximately one of four individuals referred for examination were found to have a loss of 30 db or greater at one or more frequencies in either ear.

PROCEDURE.

The general plan was to compare the percentage of teacher identifications of hearing loss with a valid criterion; namely, audiometric thresholds. Prior to the testing, each teacher was instructed to evaluate his pupils as having normal hearing or a mild-to-severe hearing loss. Each pupil was then given an individual audiometric screening test at 10 db at the octave frequencies 125-8,000 cycles and referred for pure tone threshold measurements if he failed to respond at one or more frequencies in either ear. The specific details of this procedure are to be found in an unpublished thesis by Goldman.[3] Com-

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication, March 14, 1956.

parison data were collected on 665 elementary school children from a total sample of 709 children.

RESULTS.

On the basis of the audiometric findings, the pupils were classified into two groups: those with normal hearing, and those with losses 15 db or greater. The teachers selected 53 out of the 665 for audiometric evaluation. In other words, they felt that these had hearing losses. Of the total number tested,

		AUDIOMETRIC TEST	
		% HEARING LOSS CASES	% SHOWING NO LOSS
TEACHER IDENTIFICATIONS	% HEARING LOSS CASES	a 3	b 5
	% SHOWING NO LOSS	c 16	d 76

Fig. 1. Tetrachoric table.

540 had normal thresholds. The teachers erroneously classified 33 of these as hearing loss cases. Of the 125 comprising the hearing loss group, 20 were correctly identified by the teachers. This would mean that the teachers identified approximately one out of six hearing loss cases.

For purposes of discussion, the data from the two methods are shown below in a tetrachoric table in a manner suggested by Newby[4] (see Fig. 1).

Quadrant *a* shows the percentage of agreement between the two methods in relation to the correct identification of the hearing loss cases. Quadrant *d* shows the percentage of agreement as to the normal hearing cases. Quadrant *b* shows the percentage of cases with normal hearing who were referred

by the teachers for examination. Quadrant *c* shows the percentage of hearing loss cases not identified by the teachers. To summarize, three per cent of the hearing loss cases were correctly identified by both methods. Five per cent were referred for examination but were found to have normal hearing. Sixteen per cent of the hearing loss cases were missed by the teachers. Seventy-six per cent were correctly identified by both methods as having no loss. For all practical purposes, the values in cells *a*, *b* and *c* show that teacher referrals leave much to be desired as a method of identifying hearing loss cases.

The next question raised is whether or not the teachers did better than chance. The teachers labeled 53 pupils as hearing loss cases. Of this group 33 were found to have normal hearing. Audiometric examination showed a total of 540 normals and 125 with a hearing loss. Chi square was computed and found to be 13.53, which is significant beyond the .01 level for one degree of freedom. Thus we can reject the null hypothesis of the differences being attributable to chance.

Examination of the audiometric results showed that the mean loss of those in the hearing loss group not identified by the teachers exceeded the mean loss in the identified hearing loss group by 15 db; furthermore, the six most severe hearing loss subjects were not identified by the teachers.

DISCUSSION.

The results of this study tend to support the findings of Curry, who reported that approximately one out of four hearing loss cases were correctly identified by the classroom teachers. In the present study approximately one out of six were correctly identified. The difference may stem from the criterion of hearing loss used in the present study. Since the teachers failed to detect five out of six of the hearing loss cases, it is interesting to speculate on some of the factors which may be affecting the judgments. Pupils exhibiting poor learning ability, inattention, asocial behavior, poor visual acuity, speech defects or frequent colds may be labeled as hearing loss cases. In a few instances, speech reception would be impaired sufficiently to furnish a reliable cue for identifying the

hearing loss pupil. It would seem that the teachers, although performing better than chance, were basing the majority of their judgments on factors other than hearing loss. Hearing losses of the conductive type should produce variable sensitivity as a function of time. This factor would serve to alter the composition of the hearing loss group so that a given pupil may have a loss at one time and be within normal limits at a later period, or vice versa.

It should be interesting to compare parent identification of hearing loss with those of teacher identifications. This needs further study. One might hypothesize that the parents would outscore the teachers as detectors of hearing loss.

SUMMARY.

Teacher identification of mild-to-severe hearing losses was found to be better than chance but, nevertheless, inefficient when compared to audiometric findings. In addition to detecting a low percentage of hearing loss cases this method fails adequately to recognize normal hearing. Based on these findings and those of Curry, it is recommended that audiometric examination and not teacher identification be used to detect hearing losses in school children.

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University of Kentucky.

ABSTRACT OF THE PROCEEDINGS
OF THE
AMERICAN LARYNGOLOGICAL ASSOCIATION
(Scientific Sessions)
May 13-14, 1956
SEIGNIORY CLUB, QUEBEC, CANADA

DR. BERNARD J. MCMAHON, President.
SAMUEL SALINGER, M.D., Abstract Editor.

HUMIDITY, A PROBLEM IN AIR CONDITIONING.

ARTHUR W. PROETZ, M.D.,
St. Louis, Mo.

To provide adequate relative humidity in the dwelling or office, to satisfy the needs of the nasal mucosa, is a problem that is not easily solved. With the optimum being between 40 and 45 per cent, one finds that central heating usually depresses the humidity to a point at which the mucous blanket becomes scanty and viscid, and ciliary action is slowed down. Figures assembled by Proetz from 19 cities during the month of January reveal an average outdoor humidity of 70 per cent and indoor humidity of 23 per cent. Even though the humidity can be raised to the necessary level within 30 minutes to two-and-a-half hours, depending on the apparatus employed, it would soon fall rapidly, due to losses through open doors, window cracks and even the walls themselves; consequently, the humidifying agent has to be kept going. Pans of water on the table or radiator are worthless. Commercial steam or cold vaporizers are somewhat better but not so effective as a teakettle on a gas flame.

As to humidity indicators, the sling psychrometer has been found to be the most reliable.

Central furnace type humidifiers have certain drawbacks arising from the dust which is scattered throughout the house. Unless means can be found for filtering the water this type of vaporizer leaves something to be desired.

DISCUSSION.

Dr. Lawrence R. Boies commented on the amount of valuable data assembled by Dr. Proetz on a very important subject. Although a practical solution of the problem of humidity control is still unavailable, it is comforting to know that the effort will continue to be made. Raising the humidity for therapeutic purposes has been satisfactorily accomplished in the management of acute laryngotracheitis in children. Dr. Boies has also found it practical to employ a humidifier in the postoperative care of patients whose nasal passages have been packed for a period following surgery; thus it is shown that humidity can be controlled in restricted areas in the care of the sick. If this could be extended satisfactorily to the home and the environment where people work, much would be accomplished in the prevention of the common cold and respiratory affections.

Dr. Edwin N. Broyles called attention to the plight of laryngectomy patients who suffer from dried mucus and crusting in the trachea during the winter months only. This proves the point brought out by Dr. Proetz as to the necessity of maintaining adequate humidity in the environment.

Dr. Thomas C. Galloway mentioned that low humidity in the Rocky Mountain area is well tolerated, whereas in the Middle West the same degree of humidity in the home is unhealthy. The explanation for this phenomenon may be in the dust factor which is present in the dry house. He inquired as to whether Dr. Proetz had an opinion on this point.

Dr. Proetz, in closing, offered as his explanation of the point brought up by Dr. Galloway that the distribution of pathogenic bacteria in the higher altitudes is much lower than lower down, and cited Grenfel's expeditions into Labrador, where there were no colds among the natives during the season when no ships arrived. Promptly, however, after these newcomers were landed, a severe epidemic of colds broke out among the natives with a number of deaths due, in all probability, to their inability to handle the bacterial invaders.

Dr. Proetz mentioned a very simple indication of too low humidity in a room, namely, the drawing of sparks when contacting objects.

HISTAMINE-RELEASING DRUGS.

F. C. McINTOSH, M.D. (By Invitation).

The human body contains approximately 300 mgm. of histamine concentrated mostly in mast cells along with heparin. Its action is threefold, namely, contraction of smooth muscle, increased gland activity and heightened capillary

permeability. A wide variety of drugs is listed, which are to greater or less degree histamine liberators. These include quaternary salts, morphine and other alkaloids, chemotherapeutic agents, circulatory drugs, sympathomimetic drugs, protein hydrolysates, basic amino acids and others. They seem to act through their affinity for the heparin stored within the mast cells. When a sufficient concentration has been reached the histamine-containing granules in the cells are lysed.

The relationship between the allergic or anaphylactic release of histamine and the release of histamine by drugs is a matter that has as yet not been satisfactorily explained. The author speculates that in the allergic reactions the antigen-antibody reaction may trigger some enzymic reaction leading to the formation of basic or polypeptide histamine liberators. "It is conceivable that by the local application of a histamine liberator a sensitized tissue could be depleted of its histamine stores for a long period, for instance the season of a particular pollen; the antigen-antibody reaction to the allergen could then still take place but would have fewer unpleasant consequences. The sudden unloading of the histamine depots would produce a severe reaction, but this could presumably be controlled with antihistamines and adrenalin."

The investigations conducted by the author and others are doing much to explain untoward reactions to certain drugs, and point a way to avoid or overcome them.

DISCUSSION.

Dr. Aubrey G. Rawlins reviewed the facts about histamine, its locale within the body and its physiologic action. One striking characteristic of histamine is its potentiality as a powerful endogenous agent capable of producing drastic physiologic and pathologic effects, yet under ordinary circumstances devoid of physiologic significance. Dr. Rawlins commented on the versatility of the mast cell and the various functions attributed to it by physiologists, such as its response to stress, its role in the production of ground substance of the connective tissue, its heparin-like polysaccharide which may act as an inhibitor of hyaluronidase, and now the storage and release of histamine as described by the essayist.

He submitted the question as to whether antihistamine drugs act as anti-hyaluronidase factors and whether hyaluronidase as well as histamine is liberated in cell damage, and plays an active part in inflammation; also, will hyaluronidase liberate histamine?

Dr. Sam E. Roberts inquired as to whether there is any synergistic action between heparin and histamine when given concurrently by intravenous infusion or are they antagonistic?

Dr. McIntosh replied to Dr. Rawlins' question to the effect that he knew of no evidence either for or against the activation of hyaluronidase in mast cell disintegration. In general mast cells do not survive very long in any real inflammation, although in mild chronic injuries they tend to increase in numbers. Histamine release is not of any importance in the maintenance of inflammation, because this release occurs explosively if at all, and it takes a long time for the histamine to be put back in the tissues. The duration of action of released histamine is so extremely short that the whole thing is over and done with in hours at the most, and cannot happen again for a long time.

It is possible that hyaluronidase is activated as a result of mast cell disintegration and is a subject worthy of further investigation.

As to Dr. Roberts' question, there is no appreciable synergism or antagonism between histamine and heparin; however, the histamine liberators form an insoluble complex with heparin, and heparin is a moderately active antagonist to the histamine liberators. It would be desirable if one could find a heparin analogue that might be more effective in this respect and still not have too much effect on blood clotting. Thus far such an ideal compound has not been evolved.

OTOLARYNGOLOGIC ASPECTS OF HYPOMETABOLISM.

JEROME A. HILGER, M.D.,

St. Paul, Minn.

Hypothyroidism in various degrees may be the basic etiologic factor in a wide variety of otolaryngologic conditions. The following are a few in which thyroid therapy has proven

successful as demonstrated in typical cases cited: Chronic vocal cord edema, post-thyroidectomy paresis and edema, chronic nasal obstruction with discharge, tubal catarrh, recurring serous otitis media, mucoproteinous edema of pharyngeal tissues, vascular head pains, and endolymphatic disorders. The diagnosis is reached through a careful history and proper laboratory investigation. Since the BMR test is not always reliable, it has been found that the radioactive iodine uptake is more dependable. Values of 5 per cent and below are definite indications of hypothyroidism. Where available the most accurate test is the determination of the serum protein-bound iodine.

Emphasis is laid on the importance of bearing in mind the possibility of thyroid deficiency in many of our otolaryngologic conditions where multiplicity of symptoms and chronicity and resistance to routine therapeutic procedures are dominant features.

DISCUSSION.

Dr. Francis W. Davison supported Dr. Hilger's thesis by citing three pertinent cases. One patient demonstrated the fact that hypothyroid patients can be quite thin. In this case, hoarseness, nasal obstruction and perception deafness were all due to low metabolism. The PBI was 2.34 mgm. as against a normal of 5.3. Two grains of thyroid extract daily brought relief in about three weeks. In another case hearing difficulty, due to middle ear effusion, yielded to thyroid therapy despite the fact that the BMR was only —6.

Another patient who was obese also had recurring hearing loss due to middle ear effusion. This patient was followed for 12 years and always suffered a recurrence of the ear complaint when she discontinued the thyroid.

Dr. Davison also noted that subthyroid patients usually like a room that is superheated.

Dr. Lewis F. Morrison called attention to the semantic aspect of this matter, since there is a difference between hypothyroidism and hypometabolism. It is erroneous to assume that normal limits are between —10 and +10. This was proven in an experience with patients who were hospitalized for five days with BMR of —4 or —3, but who after several days showed —23 to —35. Dr. Morrison is opposed to the empiric prescription of supplemental thyroid therapy and warns against this practice without suitable laboratory guidance.

He asked Dr. Hilger why the serum cholesterol determination was not mentioned.

Dr. James M. Robb suggested that thyroid therapy be under the supervision of the otolaryngologist rather than the internist, since the former is in a better position to evaluate the status of the auditory apparatus, as well as the nasal passages and the larynx.

Dr. Hilger, in closing the discussion, pointed out that these patients are not putty-faced individuals as a rule. The PBI determination is a satisfactory guide to therapy, and should be followed because there is no dogmatic rule governing the optimum dose for each individual.

He agreed with Dr. Morrison that the BMR is inadequate and misleading, and frequently poorly done. If facilities are not available for a PBI determination it is a simple matter to send a specimen of the patient's blood plasma to any number of well-equipped laboratories and obtain a reading in a matter of a few days.

The matter of serum cholesterol determination was purposely omitted because it is only additive information, and the day was too nice to be spent in superfluities! It is merely a confirmatory test to the BMR and if reported normal leaves one unconvinced.

Dr. Hilger appreciated Dr. Robb's suggestion that the paper be read. In this instance one could come abreast in a short time of what took him much longer to assemble. The endocrine system is amazingly important in our field, much more so than in internal medicine. Otolaryngologists have the opportunity of making the primary diagnosis more frequently than in any other branch of medicine.

The point brought up by Dr. Morrison relative to the distinction between hypothyroidism and hypometabolism is well taken. He deliberately chose the title of hypometabolism and attempted to show that there is a hypothyroid hypometabolism or a thyrotic hypometabolism, but also a hypopituitary and a hypothalamic hypometabolism, as well as the hypothyroid type. Hypometabolism may be related to hypothyroidism, but all hypometabolism is not due primarily to hypothyroidism.

FUNCTIONAL DISEASES OF THE NOSE.

STEWART NASH, M.D.,

Rochester, N. Y.

UNUSUAL VIRUS DISEASES IN OTOLARYNGOLOGY.

P. E. IRELAND, M.D.,

and

T. J. MOLONEY, M.D.,

Toronto, Can.

Virus disease seems to be on the increase and presents a wide variety of manifestations, a number of which are classified by the authors. More in detail and relating particularly to the field of otolaryngology are APC viruses, cat scratch fever and atypical pneumonias. The adenoidal-pharyngeal-

conjunctival virus was found to be mildly epidemic. In a Toronto epidemic apparently originating and being spread from swimming pools, a virus was recovered in several cases which was grown in tissue cultures of monkey-kidney epithelium or "Hela" cells.

Several cases of cat scratch fever were described, and emphasis was laid on differentiating the condition from mononucleosis, tuberculosis, Hodgkins' disease, tularemia, rheumatic fever, etc. The cat scratch skin test, which is done by injecting .1 cc of an antigen of known potency intradermally and noting the reaction after 48 hours, a positive reaction being the appearance of at least 5 mm. in diameter with an erythema of at least 10 mm. in diameter. The clinical course of the disease is described and typical cases cited.

The authors found an increased incidence of atypical pneumonias in areas where poultry raising was a prominent industry. Pigeons and turkeys were very abundant in a certain five-mile radius where these virus pneumonias were prevalent. It is hoped that further studies may identify the virus in turkeys and prove its transmission to humans.

THE VIRUS THEORY OF NASAL POLYP ETIOLOGY AND ITS PRACTICAL APPLICATIONS.

FRANCIS L. WEILLE, M.D.,
and
ROBERT S. GOHD, Ph. D.,
Boston, Mass.

In an attempt to prove that a virus is responsible for the development and recurrence of nasal polypi, Weille and Gohn conducted a number of experiments in which polypi fluid was injected into the allantoic fluid of the chick embryo. This was found to result in increased proteins in the fluid. Passage of this fluid through succesive chick embryos produced the same changes through five successive passages, although

a diminution in the virulence was noted beyond that point. This would seem to support the thesis that the infective agent was transmissible; however, in order to fulfil the last of Koch's postulates, namely to reproduce the disease in susceptible animals, attempts were made to produce gross or microscopic tissue changes in the nasal mucosa of experimental animals, all of which failed; however, the presence of a specific antibody concentration in the serum of polyposis cases and its absence in controls might be considered legitimate and important evidence. Studies will continue along the lines of devising a serological test to prove its existence.

The theory of virus etiology in polyposis, if fully proven, could well explain many of the manifestations of polyp growth, recurrence and frequent disappearance following diathermy to the stumps suggesting destruction of the intracellular viral agent.

DISCUSSION.

Dr. Lewis F. Morrison found that the common ground for discussing these two papers lay in the term "virus." He noted that no mention was made of the possible role of a virus in the etiology of laryngeal papillomata. He could not quite agree with Dr. Ireland about our present knowledge of viruses, which even in the opinion of virologists is quite meager. The points brought out with reference to the role of a virus in upper respiratory affections, adenoidal, conjunctivo-adenoidal, and the probable etiologic connection with the swimming pool are well taken.

Concerning the intradermal antigen test, there is a possibility always that the virus may not be sufficiently attenuated. Possibly the essayist may have more to say on this point.

The matter of turkey droppings as a contaminent in an area with a high incidence of atypical pneumonia seems to be a very practical consideration.

As to Dr. Weille's theory, it sounds convincing but perhaps only because nobody is in a position to refute it intelligently. Dr. Morrison felt that he could not quite divorce himself from the conviction that the allergic factors are more important than the virus. Since polyps are found in the mucosa of other organs than the nose, it would appear difficult to apply Dr. Weille's theory widely enough to cover all situations. As to the disappearance of polyps following post-excision diathermy's being due to heat killing the virus, it must be remembered that any tissue insult may be a factor in remission.

Fundamentally, Dr. Weille's work is most commendable. It is regrettable, however, that he was not rewarded by the discovery of a specific virus that could be readily identified.

He asked Dr. Weille whether the allantoic fluid had been centrifuged and examined under the electron microscope for the possible indentification of a virus.

Dr. Morrison called attention to the analogy between laryngeal papilloma and rabbit papilloma, in which a large virus was identified which is sensitive to tetracyclaine and terramycin.

Dr. Ireland replied to Dr. Morrison's query bearing on the potency of the antigen used in the skin test. The antigens are made from material taken from the so-called postule and, since the virulence is variable, it is better to take material from several lesions. As for turkey disease, he admitted that efforts at control were disappointing because of lack of co-operation on the part of the large packing companies in control.

Dr. Ireland paid tribute to his associate in the presentation, Dr. Terence Maloney, who did a great deal of the work in its presentation.

Dr. Weille stated that in the staff meetings at the Massachusetts Eye and Ear Infirmary, the privilege of becoming as violent as one pleased was accorded to all. It can, therefore, work both ways. Concerning Dr. Morrison's comment relative to the cause and effect of diathermy in the polyp cases, he failed to offer any alternative explanation to the one submitted by himself.

As regards allergy in relation to the etiology of polyps, rather than a virus, there are many people who do not believe that allergy has anything to do with polyps. It is true that the majority do believe in this relationship, but is it not possible that the allergy creates the basic immunologic response to the virus in the same way as hemolytic streptococci? Vaccines are still in vogue at certain centers, and there are those who are convinced that the immunologic responses to viruses are similar to those of the ordinary bacteria.

As to the use of the electron microscope, Dr. Weille's virologist has one available whenever he feels that it is necessary; however, up to the present he has not deemed it essential.

Mention was made of skin tests. These were tried, but without results. Since it is assumed that the antibody is the reagent resident in the skin mass, it is possible that a more thorough investigation along the line of the alphibetical APC. notation by Hubner and other workers might evolve a dependable serologic test; also if a few volunteers could be found it might be possible to produce polypi in the nose experimentally.

MANAGEMENT OF NON-MALIGNANT GROWTHS IN THE MAXILLARY SINUS.

O. E. VAN ALYEA, M.D.,

Chicago, Ill.

Polypi and cysts are the most common non-malignant growths found in the antrum. The former develop most often in the vicinity of the ostium as a result of irritation, which may be an allergy or a pathogen from infection. They may grow into the sinus cavity or out of it through the ostium,

appearing in the middle meatus or even growing backward into the choana.

Cysts may be either the simple mucous type or may originate from the dental appendanges in the form of radicular or follicular cysts. Frequently they are symptomless but at times may be responsible for local numbness, nasal congestion and frontal headaches.

Diagnosis in all cases can readily be made through the use of contrast media with X-rays. Most cysts are found in the floor of the antrum or the lateral walls, and are usually smooth and almost spherical in outline. Polypi, on the other hand, are more irregular in outline and most common along the vaso-antral wall. If they cannot be controlled by antiallergic therapy they should be removed via a sublabial approach. The antrum mucosa is in most cases left undisturbed.

Cysts may at times be eradicated through an intranasal antrostomy, although in many instances it is necessary to resort to the Caldwell Luc operation because of their location and size.

DISCUSSION.

Dr. Robert Goodale emphasized the point that cysts of the antrum are not to be ignored or minimized, particularly if the patient has symptoms referable to the one side. He cited a case of uveitis which failed to respond to therapy until an apparently simple cyst of the antrum was removed, revealing pus and a predominantly pneumococcus infection. Too much reliance should not be placed on the antibiotics when infection is present along with a cyst.

Polypi and cysts are common findings. Dr. Goodale favors the removal of polypi developing within the antrum via the canine approach. It is not necessary to remove the mucosa entirely. The surgery should be limited to the presenting pathology. Additional drainage through an inferior meatus opening is not always necessary. He agrees with Dr. Van Alyea that the possibility of malignancy in all these cases should never be overlooked. All material removed should be sent to the pathologist.

Dr. Lewis F. Morrison inquired as to the presence of organisms in the cysts. Previous reports on this item have been contradictory.

Dr. Robert S. Priest wanted to know whether Dr. Van Alyea feels that surgical exploration is indicated in all cases of cysts. He has had the experience of passing up some of these benign growths only to have the patient return complaining of pain, and subsequent biopsy revealed mixed tumor.

Dr. Van Alyea replied to Dr. Morrison's query by stating that cultures have been done routinely, but that he had no significant report to

offer; besides, in many cases the cyst was opened via the intranasal route, often merely as a result of antrum irrigation.

EXPERIMENTAL TREATMENT OF RECURRENT CARCINOMA OF THE NASOPHARYNX WITH ELECTRODESSICATION, RADIOACTIVE COBALT AND X-RAY RADIATION.

FRANCIS A. SOOY, M. D.,

San Francisco, Calif.

Six cases of recurring inoperable carcinoma of the nasopharynx previously unsuccessfully treated with irradiation were reported by Dr. Sooy. These were treated by electrodessication and local intranasal radiation with radioactive cobalt. The greater part of the septum was removed to afford more space for the transnasal manipulations. Three of the patients were alive and free from signs of recurrence more than six years following therapy. Subsequently ten additional cases were similarly treated, with five of them surviving for periods of from six months to four years.

Cases showing clinical or X-ray evidence of extension into the base of the skull were excluded.

The electrodessication was carried out with caution, lest excessive heat cause damage to the pituitary and optic nerves.

The radioactive cobalt was applied at 1 cm. distance from the area, via a Foley urethral catheter, the bag being distended with radio-opaque solution to permit accurate placement of the cobalt. The dosage administered was 4000 to 6000 r.

Although considerable dryness was observed, there was very little crusting after the initial slough and sequestra had been removed.

DISCUSSION.

Dr. Harry P. Schenck felt that this presentation teaches us not to be too pessimistic about some of these malignancies. He has always

felt that electrocoagulation was not employed widely enough in otolaryngological practice. One reason may be the fact that local anesthesia often limits the extent and degree of the coagulation as compared to the freedom afforded under general anesthesia.

Concerning the freedom from crusting following removal of the septum reported by the author, Dr. Schenck feels that there must be some difference in the influence of climate, because the cases he has seen in Philadelphia manifest marked crusting for months.

The low incidence of metastases in the cases reported is rather striking in view of the size of the primary lesions. In his cases, he has not been so fortunate.

The advantage of the technique described is obvious since it affords a wide approach to all the affected area.

In this connection, Dr. Schenck suggested that this procedure need not be restricted to carcinoma alone. He had had a good result in an extensive chordoma with destruction of the sella turcica by wide electrocoagulation applied through both sides of the nose as well as behind the palate. He did not destroy the septum.

Dr. Thomas C. Galloway has for many years been enthusiastic about the use of electrocoagulation.

It is important to know something about the character of the tumor, many of them being lympho-epitheliomas or transitional cell carcinomas, both very malignant with early involvement of the glands. He noticed that the authors used X-ray therapy in some of these cases and wondered how much of the good result might be attributed to this medium rather than the cobalt. Removing the septum to afford an approach to the tumor impressed him like Lamb's story about the Chinaman who burned a house down when he wanted a roast pig.

He had found it practical to use Dr. Mosher's old incision, modified by a trapdoor through which the carcinoma involving the frontals, antrums and sphenoids could be completely visualized and destroyed; also at times it is just as feasible to approach the area via a transpalatal incision.

It was his impression that tumors arising from the septum were usually of the squamous cell type, being less malignant and more effectively destroyed by a less mutilating procedure.

The outlook for the more malignant types is not so dark as previously considered. He has had the good fortune of seeing some of these cases cured in recent years and considers the combined use of electrosurgery and radiation as being responsible. He assumes that the essayists were using the biterminal electrodes which penetrate more deeply than the superficial dessicating current.

Dr. Sooy replied that their dessicating unit is adjusted to achieve a slough of about 7 cm. of bone and mucosa.

The transpalatal approach was considered but rejected in favor of the one described, because they wanted to preserve a wide aperture for inspection of the site in the postoperative period.

In addition to the symptoms of nasal obstruction and bleeding and cervical metastases, they frequently found early indication of the growth in the ear symptoms such as unresolved serous otitis media. In three cases a prophylactic biopsy in Rosenmuller's fossa yielded positive evidence of lymphadenoma.

CILIARY STREAMING THROUGH THE LARYNX AND DISTRIBUTION OF LARYNGEAL EPITHELIUM.

A.C. HILDING, M.D.,

Duluth, Minn.

Studies were made on the distribution of squamous and ciliated epithelium in five human larynges removed at autopsy and in a number of recently killed calves. The results corresponded to the generally accepted pattern except that above the vocal cords the squamous epithelium is apt to occur in islands or peninsulas, surrounded by ciliated columnar epithelium. Further observations on the patterns of ciliary streaming seemed to indicate two streams coming up from the trachea. The posterior one passes between the arytenoids to the rim of the larynx. The anterior stream is split into two parts at the squamous cell barrier of the cords, flowing backward beneath the cord to the arytenoids.

The absence of ciliary currents in certain areas of the vocal cords seems to suggest a predisposing factor in the occurrence of carcinoma on the cords in 80 to 85 per cent of all malignancies of the larynx. Whether the inhaling of cigarette smoke or other noxious vapors in the atmosphere is as important etiologic factor as other factors such as vocal trauma, etc., is a matter that requires further study and analysis.

DISCUSSION.

Dr. G. Edward Tremble mentioned the statement found in most standard textbooks that the mucous membrane of the human larynx varies according to the functions performed; thus the difference between the squamous epithelium of the true cords and the ciliated columnar types with goblet cells in other areas. It is important to remember that the interarytenoid space contains both types of epithelium. When the arytenoids approximate, the tissue between them is thrown into folds, the ridges being covered with squamous epithelium and the valleys with columnar ciliated. Nature has provided a similar arrangement in the nasal mucosa. Dr. Tremble described the ciliary streams coming up the trachea, the posterior stream going directly to the interarytenoid space and the anterior passing below the cords to reach the same area. Convergence of the streams at this point probably explains the high incidence of tuberculosis lesions in the posterior portion of the larynx.

Dr. Tremble has used dicalcium phosphate powder which turns bright orange color when in contact with moisture and serves as a good guide

in determining ciliary streaming. The powder thus applied in the trachea seems to follow a spiral rather than a straight course, which may be due to the movements of the trachea in respiration.

Dr. Arthur W. Proetz was impressed by the finding described by Dr. Hilding where the ciliary stream causes material to accumulate in the center just below the cords before spreading and passing posteriorly. This is just the area where subglottic lesions develop which are frequently overlooked, however, it must be noted that in the specimens observed by Dr. Hilding, the cough phenomenon was absent, of course, so that one cannot precisely evaluate the results. He would like to see this part of the investigation carried on with this reflex in mind.

The point brought out by Dr. Hilding regarding the mucous anchors of thick mucus coming out of the glands, and their influence in retarding the movement of the mucous blanket, ties in with his own observations on the effects of altered viscosity on this function, often the result of inadequate humidity and at times the result of limited thyroid activity, as pointed out by Dr. Hilger.

Dr. Proetz was wondering what happened to the spiral phenomenon described by Dr. Franklin, of London, and just mentioned by Dr. Tremble. It calls to mind the beautiful demonstration of spiral streaming in the sinuses which Dr. Hilding demonstrated some time ago.

Dr. Thomas C. Galloway inquired how fixed and persistent is the pattern of squamous and ciliated epithelium, and how much metaplasia occurs as a result of trauma, chemical irritation, etc.

Dr. Hilding called attention to differing predilection in sites between tuberculosis, which occurs more frequently in the posterior portion of the larynx, whereas carcinoma was more common in the anterior. The facts are still not entirely clear.

Since these experiments were performed in dead animals, the findings would differ from those in the living because of the absence of the respiratory movements and changes in dimensions. Spiral streaming was not observed in these animals; however, in one human trachea which he obtained some months ago there was a very definite spiral flow.

Regarding the question brought up by Dr. Galloway, from data obtained in similar experiments in the nose, he could only infer that the pattern of epithelium changes according to the stresses, normal and pathologic, which occur or are induced in the areas under observation.

* * *

Dr. John A. Murtagh introduced Dr. Campbell, guest speaker, with whom he had been associated for ten years in a study of the basic factors of the larynx and recurrent laryngeal nerve, and expressed his appreciation of his valuable contributions to the knowledge of the subject.

ELECTRICAL MANIFESTATIONS OF RECURRENT NERVE FUNCTION.

C. J. CAMPBELL, M.D.,
and
JOHN A. MURTAGH, M. D.,
Hanover, N. H.

A number of experiments were conducted on goats to determine the presence of mixed components in the recurrent laryngeal nerves. These nerves were dissected free in the anesthetized animals. "Stimulating electrodes connected to a Gross square stimulator and recording electrodes leading to a pre-amplifier, amplifier and cathode ray oscillator were placed on the nerve."

The nerve was crushed distally, and the recordings of repetitive stimulation, as traced in the oscilloscope, photographed on a drawing board for enlarged tracings.

Much of the information thus gleaned pointed to the existence of proprioceptive afferent nerve elements from the laryngeal musculature. These investigations would seem to support the contention of Sherrington that motor nerves possess afferent as well as efferent fibers. The reciprocal inhibition shown by antagonistic laryngeal muscles adds further weight to this thesis.

The recordings were made at the peripheral end of the nerve, thus eliminating any influence from the tracheal and esophageal branches.

The experiments also appeared to demonstrate a double nerve supply to the cricothyroid muscle (superior laryngeal and recurrent).

DISCUSSION.

Dr. Louis H. Clerf speculated on the question as to how far one can translate findings in experimental animals to the human being. A case in point is the finding by one investigator of motor fibers distributed to the arytenoid muscle from the superior laryngeal nerve in man, whereas other investigators, failing to find these fibers in the dog, assumed the same to be true in man. It is of interest that pressure

applied briefly to the recurrent nerve does not interfere with its conductivity; however, he suspected that remote effects would arise as shown in several patients operated upon for pulsion diverticulum who developed unilateral paralysis apparently due to pressure of metallic retractors applied for 10 to 15 minutes.

Dr. Clerf was in agreement that one cannot rule out reflexes as a possible explanation of some of the results obtained in these and similar studies. They demonstrate that the degree of stimulation employed greatly influences the reactions, the interpretation of which is taken into account in the final analysis of the results.

While the question of proprioceptive elements in the muscles of the larynx has not been proven, having in mind the remarkable reciprocal relationship of the external ocular musculature and its similarity to the two halves of the larynx, it would appear that further study of the end organs in the laryngeal musculature is necessary.

Dr. Lewis F. Morrison stated that in view of the variable types and numbers of bifurcations of the nerve in the neck, it might be of value to do the laryngectomies from above downward rather than the reverse; also some knowledge might be gained by dusting some powder in the trachea and following its course while the procedure is being carried out.

He inquired of the essayists as to the formula for the amount of voltage employed and the duration of its application.

Dr. Campbell replied that if the facilities were adequate one might well repeat the experiment of Ogura and Aulenton in the superior laryngeal nerve stimulated *in situ* and following laryngectomy using the extirpated nerve as they have done in the goat.

He believed that it is necessary to have square wave stimulators, the Grace instrument being the type he employed. Stimulus applied for one-tenth of a millisecond at one to three volts should be sufficient for most of the fibers; however, if one is desirous of observing the individual muscles the whole gamut of strength and duration would have to be used to get anything worth while.

Reverting to Dr. Clerf's point about the pressure, in his experiments it was applied for only a few minutes. Others have used pressure for longer periods and obtained a blockade of the function of the larger fibers. Lexal devised an instrument which would block out the largest fibers but retained function of the slower ones in the Gamma group, but finally decided that just pinching the nerve between the thumb and finger for 30 to 60 minutes would accomplish the same thing. The time element is important, and it may be that significant data may have been missed by not carrying the experiment over a longer period of time.

Referring to another item mentioned by Dr. Clerf, he did attempt to examine the muscles to see if these were spindles present, which was not the case; however, they did take the precaution of examining the muscles, in which everyone agrees that they are present. So the difficulty must have been with them and their present techniques, which he hoped would soon be improved.

SUBMUCOSAL COMPARTMENTATION OF THE LARYNX.

JOEL J. PRESSMAN, M.D.,

Beverly Hills, Calif.

The subject of this presentation was suggested to the author in his studies of the fate of radioisotopes injected into the larynx of experimental animals. It resulted in the observation that the submucosal area was divided into a number of compartments. Fluid injected in these spaces failed to pass from one to the other. Dyes were injected into certain areas of the larynx during the course of a laryngectomy and the distribution studied at the conclusion of the operation. Numerous colored slides were shown which demonstrated that the dye failed to cross the midline from one compartment to its opposite fellow. This applied to the epiglottis, vocal cords, ventricular bands and arytenoids. The same results were obtained in similar experiments on the living dog. The direction of the spreading dye was studied in a variety of situations and demonstrated the areas and extent of the various compartments. These findings would seem to coincide with the results of Dr. Hilding's experiments on ciliary flow, and also are in line with the distribution and direction of the lymphatics. They also suggest an explanation of the method of spread of carcinomas arriving in various locations within the larynx.

DISCUSSION.

Dr. Paul H. Holinger brought up the point about the role of the small holes in the cartilage of the epiglottis as an avenue of escape for the fluid from one compartment to another, a matter that would have to be determined by the microscope rather than by gross appearance, in view of their tiny caliber.

In some of the slides shown it appeared to him that the fluid did cross the midline, and he wondered whether it was due to the lymphatic distribution or perhaps an intracellular pickup. He thought the compartments might be better delineated with fluid injected under a lesser degree of pressure. Considering the clinical interpretation of edema of the larynx, its favorite sites and manner and spread, this is all understandable on the basis of anatomy, the phonating margin of the cord being tightly bound down and the subglottic area being loosely areolar; also it is conceivable that the direction of the various muscles and their compartments may have much to do with the distribution of injected fluids.

It is of vital importance to recognize the role of the lymphatics in the spread of carcinoma in the larynx, which predispose certain areas to a carry-over from one side to the other.

Dr. Chevalier L. Jackson considered this presentation as another example of a new anatomical concept having considerable clinical significance. He was impressed by its analogy to the subject of segmentation of the lung, in which he has been deeply interested for some time. The clinical application of this newer knowledge of anatomy has had a direct bearing on the surgery of this organ; however, in the matter of clinical application of Dr. Pressman's findings to the question of carcinoma of the larynx, it would be premature to apply them too closely at this time, although the possibilities in other directions are quite evident.

Dr. Pressman agreed with Dr. Jackson in the matter of cancer in relationship to his experiments; nevertheless, it is a subject to which some attention should be directed. Dr. Holinger's point about the spread of the dyes across the midline has merit, because we know that structures such as nerves and vessels do pass from one side to the other, thus affording possible channels for spread. It should be remembered, however, that the degree of pressure and the amount of fluid injected would have much to do with either the limitation within the compartment or its forcible spill-over into the opposite side.

This study has at least demonstrated to him in his work with radioisotopes that a mass injection in one space will not accomplish the results obtained when several spaces are injected.

CANCER OF THE LARYNX.

FERNAND MONTREUIL, M.D., (By Invitation).

Analysis of a series of 70 cases of carcinoma of the larynx covering a period of three-and-a-half years was presented by Dr. Montreuil. The cases were divided into three groups: intrinsic (confined to the vocal cord); extrinsic (within the laryngeal box); and extra-laryngeal. It was pointed out that the symptoms were more dependent upon the site of the growth than its size and extent. For instance, vocal cord lesions produce hoarseness in the earliest stages when the lesion is small, whereas in larger lesions away from the cords, the disturbance of the voice may be delayed.

Direct laryngoscopy and biopsy are essential for diagnosis.

Because of the unpredictability of the virulence of various lesions, it is considered advisable to do a neck dissection in all cases of extrinsic and extra-laryngeal lesions.

The author's experience with laryngectomy after extensive irradiation was not good, the wounds breaking down early with delayed healing.

Laryngectomy is usually done under local anesthesia except when preceded by tracheotomy or when neck dissection is contemplated. The T-incision is preferred, with removal of the hyoid bone and strap muscles. Early ambulation is encouraged.

DISCUSSION.

Dr. Henry B. Orton prefers to classify his cases according to the anatomical site of its origin. A record of a series of cases which he presented at a recent meeting of the Academy showed that his best results following surgery were in the following order: Cords, epiglottis, ventricles, arytenoid, subglottic, epiglottis and tongue, ary-epiglottic fold and pyriform sinus.

Dr. Orton called attention to one area frequently overlooked, namely, the vallecula, where exposure may be difficult if the epiglottis is close to the tongue. In these cases a transhyoid pharyngotomy will often spare the patient a total laryngectomy. Dr. Orton prefers general anesthesia, because there is less shock to the patient when he realizes he can no longer talk. Also he prefers a "U" incision, which affords better exposure.

Dr. Montreuil's operative mortality of 4 per cent seems a little high in view of his own experience in 450 cases with a mortality of only 1.3 per cent.

As for radiotherapy, he is averse to its use either pre- or postoperative, because of possible complications such as radionecrosis.

Dr. Francis E. LeJeune was interested in one item touched upon by Dr. Montreuil, namely, the possibility of implanting the carcinoma lower down in the bronchiopulmonary tract by way of the diagnostic bronchoscope. In this connection it is worth while mentioning the respect with which general surgeons treat the primary lesion, taking care that the knife and frequently the gloves used for the incision are discarded in favor of fresh ones.

Dr. Edwin N. Broyles inquired as to why the author fed his patients by intravenous injection rather than via an esophageal tube.

Dr. Montreuil agreed with Dr. Orton that various classifications of laryngeal carcinomas are fequently puzzling. It has been his policy to describe the lesion according to its exact location.

Concerning his reluctance to use a feeding tube, he was probably influenced by the fact that it causes the patient considerable discomfort, both in the nose and in the pharynx. This was particularly true of the old rubber tubes; however, the newer polyethelene tubes are much less irritating and have been used by him in cases where tube feeding has to be carried on for some time because of a fistula.

BENIGN GROWTHS OF THE VOCAL CORD: CLINICAL vs. MICROSCOPIC DIAGNOSIS.

SAMUEL SALINGER, M.D.,

Chicago, Ill.

An analysis of over 250 consecutive biopsies from the vocal cords yielded some interesting data from the viewpoint of comparing the preoperative clinical diagnosis with the microscopic findings. It was shown that the tumors which were definitely neoplastic, such as carcinoma, papilloma, etc., were generally recognized by their gross appearance and were confirmed by characteristic microscopic pictures; however, in the case of non-neoplastic growths the divergence was remarkable. Dr. Salinger found fault with the diagnosis of "vocal cord polyp" because in 81 cases so diagnosed, not one was given such a designation by the pathologist. In a considerable number of this group the growth was labeled "edamatous fibroma," but the remainder covered a wide variety of conditions, which were named according to the predominant changes found in the various components of the tissue. The author is convinced that there is no characteristic histologic picture peculiar to the so-called polyp and that the term is, therefore, misleading, since it implies a definite entity, which it isn't.

DISCUSSION.

Dr. Francis E. LeJeune called attention to a description of polyp pathology in Coates, Schenck and Miller's recent "Otolaryngology," indicating a characteristic stroma of edematous tissue with prominent vascular changes. Similarly, Morrison and Eggston and Wolf found these growths to be characteristic of a traumatic or inflammatory nature with frequent thrombosis of blood vessels, which has given rise to the diagnosis often given by pathologists of "angiomatous polyp." Dr. LeJeune agreed that these are benign growths, but would prefer to retain the designation of "polyp" as a challenge to pinpoint our diagnosis.

The disparity between clinical and histological diagnosis in the 81 cases cited by Dr. Salinger is not entirely the fault of the pathologist, but should be attributed to the lack of unanimity of thought between the patholgist and the laryngologist.

Dr. James M. Robb recalled having read a paper in which he termed the controversial growth mentioned by Dr. Salinger a "hemorrhoid of the vocal cord." He was impelled to so designate it because of the re-

port of the pathologist, who said "in my opinion frequently there is a dilated vessel, a dilated blood vessel, that in time undergoes changes; chronic irritation always produces fibrous tissue which always contracts." Dr. Robb would be very glad to find some other label to attach to this condition, but because it seemed to be mainly a varix he thought "hemorrhoid" was not too bad.

Dr. Salinger was glad to have the references mentioned by Dr. LeJeune, in which the authors seemed to agree on a typical microscopic picture; however, from his own experience he is not convinced. The fact that the pathologist in examining a polyp rarely calls it a "polyp" indicates the lack of uniformity in the histologic analysis. Until pathologists can agree on a characteristic histologic picture peculiar to the polyp, he is unwilling to employ that term, and would prefer naming it simply "a benign growth of the vocal cord."

PRIMARY MALIGNANT TUMORS OF THE UVULA.

FRED Z. HAVENS, M.D.,
and
RICH C. YE, M.D.,
Rochester, Minn.

Primary malignancy of the uvula is very rare. Havens and Ye were able to collect only seven cases that were observed at the Mayo Clinic from 1919 to 1952. During the period there were some 1,500,000 patients admitted to the Clinic. In 42.8 per cent of the cases secondary lesions were present, which led to disaster. The lesion is usually an ulcer or a granulated area on the uvula which appears indurated and stiff rather than flabby, as is the normal uvula. The authors advise removing the organ by surgical diathermy. Neck resection was not carried out in cases where there was no evidence of lymph node involvement.

Only two of the cases survived, one for four years and the other for one year when last heard from.

DISCUSSION.

Dr. Leroy Schall, reviewing his records of 12 cases seen since 1935, finds himself in agreement with the essayists. He is impressed by the absence of early symptoms in some of these cases. In one instance it was discovered by the patient's dentist. Unfortunately, many of these patients come in too late, and it is difficult to determine whether the lesion was primary in the palate or in the uvula. Eight cases were

treated by electrosurgical removal; six showed no recurrence for periods of one, two, six, seven, eight and ten years. Four cases were treated by irradiation. Three showed no recurrence after three, four and six years respectively.

Dr. James M. Robb inquired whether Dr. Havens removed the soft palate. He noted that one patient was dead, but was wondering whether the others have been followed up to date. Certainly a malignant lesion of the soft palate is a serious problem.

Dr. Havens said that, like Dr. Schall, he was unable in many cases, judging by the records, to determine just where the lesion started; therefore, he omitted these cases from his series. It was his desire to point out the danger of this lesion even when it was localized to the palate alone. Thus, future investigators could be guided in their evaluation of the lesion by the knowledge that this series included only those cases in which it was positively demonstrated that they were primarily in the uvula.

Too much emphasis cannot be placed on the importance of careful examination. One case cited had passed through the hands of a very competent otolaryngologist who was very much embarrassed at having missed the lesion. In this case it involved only the very tip of the uvula, but on palpation seemed to be firm enough to excite suspicion.

Replying to Dr. Robb's question, the answer would be to take off as much tissue as necessary, even if it meant removing the entire palate.

In the one case which was operated on the lesion involved only a third of the uvula. All the others were years back.

USE OF THE LARYNX IN REPAIR OF CICATRICIAL STENOSIS OF ESOPHAGEAL INTROITUS.

JOHN B. ERICH, M.D.

Dr. Erich reported a case in which the larynx was attached to the esophagus to bypass a structure at the introitus resulting from radiation necrosis, following therapy for a carcinoma. The patient was a man of 68. The lesion, a large malignant tumor, Grade III, occupied the larynx and hypopharynx, causing marked obstruction; also there was an enlarged cervical node.

After tracheotomy the growth was destroyed by surgical diathermy following which radon seeds were implanted at the site of operation as well as into the lymph node. Feeding was accomplished through a tube. Marked edema and perichondritis with sequestration ensued. Eighteen months later there was no evidence of recurrence.

It was found that the vocal cords were in complete abduction, which was favorable for the technique which followed. The larynx was split and severed from the trachea, which was dissected free from the esophagus for a distance of 3 cm., and its free margin then sewed to the skin. The esophagus was severed just below the stricture and attached to the mucosa of the larynx, which then served as its upper end. Since the cords were well abducted, soft food passed readily, and within a short time the patient was able to swallow any type of well-masticated food. Three years have elapsed, and the condition is satisfactory, with no indication of any recurrence.

MECHANICAL APPLIANCE FOR CICATRICIAL STENOSIS OF LARYNX AND UPPER PART OF TRACHEA: REPORT OF A CASE.

JOHN B. ERICH, M.D.

Stenosis of the larynx, due to perichondritis following an automobile accident in a man of 26, was treated by excising of the scar tissue through a thyrotomy and the implanting of a Thiersch graft over a sponge. After the graft had taken, an acrylic obturator was prepared and worn by the patient. At the time of operation it was noted that the esophageal wall protruded into the lumen of the larynx due to loss of a great portion of the cricoid cartilage. A small fistula developed which failed to heal, despite closure with a flap of mucous membrane. It was then necessary to cover the area with skin, which was implanted via a tube flap obtained from the clavicular area, and brought up into the larynx. The fistula was thus closed, but the redundancy of the skin flap so narrowed the lumen as to prevent adequate breathing. Consequently, an acrylic tube was fashioned with a ring-shaped extension protruding through the tracheostomy, through which the tracheal cannula could be inserted. A hole in the tip of the cannula permitted air to pass from the trachea into the upper air passages so that breathing through the nose and mouth could be accomplished when the external orifice

of the cannula was corked. The cannula and acrylic device could be easily removed for cleansing and has been worn with satisfaction for two years up to date.

ANNOUNCEMENTS

The American Otorhinologic Society for Plastic Surgery takes pleasure in announcing the following:

2. A course in Plastic Surgery of the Head and Neck will be given in New Orleans, Louisiana, February 17-22, 1957, at the Louisiana State University. This course is to include cadaver, didactic and wet and dry clinics.

Early registration is recommended because of the limited facilities. Judging from our previous courses there will be oversubscription.

For further information address Dr. L. J. Feit, Secretary, 66 Park Ave., New York 16, N. Y.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The meeting dates of the Sixth International Congress of Otolaryngology are again emphasized as May 5th through May 10th, 1957. The scientific program for the Plenary Sessions is now complete and is as follows:

CHRONIC SUPPURATION OF THE TEMPORAL BONE.

OPENERS: Marcus Diamant, Central County Hospital, Halmstad, Sweden—Anatomical Etiological Factors in Chronic Middle Ear Discharge.

Luzius Rüedi, Zurich, Switzerland—Pathogenesis and Treatment of Cholesteatoma in Chronic Suppuration of the Temporal Bone.

Horst Wullstein, Director, Otolaryngological Clinic, University of Würzburg, Germany—Surgical Repair for Improvement of Hearing in Chronic Otitis Media.

DISCUSSERS: A. Tumarkin, Liverpool, England; Juan Manuel Tato, Buenos Aires, Argentina; T. E. Cawthorne, London, England; Fritz Zöllner, Freiburg, Germany.

COLLAGEN DISORDERS OF THE RESPIRATORY TRACT.

OPENERS: Hans Selye, Director, Institute of Experimental Medicine and Surgery, University of Montreal, Faculty of Medicine, Montreal, Canada.

Introduction:

Michele Arslan, Padua, Italy—The Upper Respiratory Tract.

Leslie Gay, Physician-in-Charge, Allergy Clinic, The Johns Hopkins Hospital, Baltimore, U. S. A.—The Lower Respiratory Tract.

DISCUSSERS: Victor E. Negus, London, England; Branimir Gusic, Zagreb, Yugoslavia; Aubrey G. Rawlins, San Francisco, U. S. A.; Henry L. Williams, Rochester, Minn., U. S. A.

PAPILLOMA OF THE LARYNX.

OPENERS: Jo Ono, Tokyo, Japan—Etiology.

Plinio de Mattos Barretto, Faculty of Medicine, University of Sao Paulo, Brazil.

Diagnosis:

F. C. W. Capps, London, England—Therapy.

DISCUSSERS: C. A. Hamberger, Göteborg, Sweden; Pedro Hernandez Gonzalo, Havana, Cuba; Eelco Huizinga, Groningen, Netherlands; Albert von Riccabona, Vienna, Austria.

Applications to present voluntary papers are being received regularly and anyone wishing to present such a paper should make known his intentions prior to the deadline of Oct. 1, 1956.

The motion picture sessions, as well as the scientific exhibits, will prove to be outstanding in every respect. It is essential that anyone wishing to present an exhibit should make his intentions known immediately, as the final date for consideration is Aug. 1, 1956; applications to present motion picture films should be sent in before Oct. 1, 1956. Anyone planning to attend the Congress and who has not yet registered should do so immediately in order to obtain hotel registration priority.

For more detailed information pertaining to the Sixth International Congress please communicate with the General Secretary, 700 N. Michigan Ave., Chicago 11, Ill., U. S. A.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. John R. Lindsay, 950 East 59th Street, Chicago 37, Ill. ....
Vice-President: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.
Secretary-Treasurer: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Statler Hotel, Washington, D. C., May 4, 1957.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. LeRoy A. Schall, Boston, Mass.
First Vice-President: Dr. Henry M. Goodyear, Cincinnati, Ohio.
Second Vice-President: Dr. Robert E. Priest, Minneapolis, Minn.
Secretary: Dr. Harry P. Schenck, Philadelphia, Pa.
Treasurer: Dr. Fred W. Dixon, Cleveland, Ohio.
Meeting: Statler Hotel, Washington, D. C., May 3, 1957.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Percy Ireland, Toronto, Canada.
President-Elect: Dr. Lewis F. Morrison.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Statler Hotel, Washington, D. C., May, 1957.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: John R. Lindsay, M.D., Chicago, Ill.
Vice-Chairman: James W. McLaurin, M.D., Baton Rouge, La.
Secretary: Hugh A. Kuhn, M.D., Hammond, Ind.
Representative to Scientific Exhibit: Walter Heck, M.D., San Francisco, Calif.
Section Delegate: Gordon Harkness, M.D., Davenport, Iowa.
Alternate Delegate: Dean Lierle, M.D., Iowa City, Iowa.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 14-20, 1956.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Clarence W. Engler, 2323 Prospect Ave., Cleveland, Ohio.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May 21-23, 1958.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., October 8-11, 1956.

THE AMERICAN RHINOLOGIC SOCIETY.

President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. James Chessen, 1829 High St., Denver, Colo.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, Illinois, October, 1956.
Annual Meeting: Palmer House, Chicago, Illinois, October, 1956.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. D. M. Lierle, University Hospital, Iowa City, Iowa.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Palmer House, Chicago, Ill., October, 1956.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Gordon D. Hoople, 1100 East Genesee St., Syracure, N. Y.
Secretary: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Meeting: Palmer House, Chicago, Ill., October, 1956.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Joseph Gilbert, 111 E. 61st St., New York, N. Y.
Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pittsburgh, Pa.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Arnold L. Caron, 36 Pleasant St., Worchester, Mass.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Jose Gros, Havana, Cuba.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Sixth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Brazil, 1958.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.
General Secretary: Dr. Paul Holinger, 700 No. Michigan Ave., Chicago 11, Ill.
Meeting: Statler Hotel, Washington, D. C., May 5 - 10, 1957.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. Chevalier L. Jackson.
Vice-President: Dr. John J. O'Keefe.
Treasurer: Dr. Joseph P. Atkins.
Secretary: Dr. Louis E. Silcox.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. Harry P. Schenck, Dr.; Benjamin H. Shuster, Dr. William A. Leil, Dr.; William J. Hitschler.

BALTIMORE NOSE AND THROAT SOCIETY.

Chairman: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Maryland.
Secretary-Treasurer: Dr. Theodore A. Schwartz.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Dr. Raymond Kerwin, 310 So. Michigan Ave., Chicago, Ill.
Vice-President: Dr. Frank Wojniak, 6132 So. Kedzie Ave., Chicago, Ill.
Secretary-Treasurer: Dr. Stanton A. Friedberg, 122 So. Michigan Ave., Chicago, Ill.
Meeting: First Monday of each Month, October through May.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. J. L. Levine.
Vice-Chairman: Dr. Russell Page.
Secretary: Dr. James J. McFarland.
Treasurer: Dr. Edward M. O'Brien.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Frederick A. Holden, Medical Arts Building, Baltimore 1, Maryland.
Chairman-Elect: Dr. Sherman B. Forbes, 706 Franklin Street, Tampa 2, Florida.
Vice-Chairman: Dr. William J. G. Davis, 1632 K Street, N. W., Washington 6, D. C.
Secretary: Dr. G. Slaughter Fitz-Hugh, 104 East Market Street, Charlottesville, Virginia.

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Howard L. Mitchell, Lexington, Va.
President-Elect: Dr. L. Benjamin Sheppard, 301 Medical Arts Building, Richmond 19, Va.
Vice-President: Dr. Edgar Childrey, Jr., Professional Building, Richmond, Va.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Va.
Annual Meeting.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. H. K. Rouse, 1300 27th Ave., Gulfport, Miss.
Vice-President: Dr. A. J. McComiskey, 3420 Prytonia St., New Orleans, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: The Edgewater Gulf Hotel, Edgewater Park, Miss., May 17-18, 1957.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. J. C. Peele, Kinston Clinic, Kinston, N. C.
Vice-President: Dr. George E. Bradord, Winston-Salem, N. C.
Secretary-Treasurer: Dr. J. D. Stratton, 1012 Kings Drive, Charlotte 7, N. C.
Meeting:

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Vice-President: Dr. J. H. Gressette, Orangeburg, S. Car.
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Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. A. R. Hollender, 605 Lincoln Road, Miami Beach, Fla.
Vice-President: Dr. Curtis D. Benton, Jr.
Secretary: Dr. James H. Mendel, Jr., 7241 Red Road, Miami, Fla.
Meeting: Quarterly, at Seven Seas Restaurant, February, May, October, and December.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Edwin G. Grafton.
Vice-President: Dr. L. A. Furchgott.
Secretary-Treasurer: Dr. Hal W. Maxwell.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Leland R. House, M.D.
Secretary-Treasurer: Sol Rome, M.D.
Chairman of Opthalmology Section: Stephen J. Popovich, M.D.
Secretary of Ophthalmology Section: Richard Kratz, M.D.
Chairman of Otolaryngology Section: Harold Boyd, M.D.
Secretary of Otolaryngology Section: Howard G. Gottschalk, M.D.
Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles, California.
Time: 6:00 P.M., first Monday of each month from September to June inclusive—Otolaryngology Section. 6:00 P. M. first Thursday of each month from September to June inclusive—Ophthalmology Section.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: H. Leroy Goss, M.D., 620 Cobb Bldg., Seattle 1, Washington.
Secretary-Treasurer: Homer E. Smith, M.D., 508 East South Temple, Salt Lake City, Utah.
Meeting: April 7-11, 1957.

THE SOCIETY OF MILITARY OTOLARYNGOLOGISTS.

President: Col. Wendell A. Weller.
Secretary-Treasurer: Major Stanley H. Bear, M.C., 3810th USAF Hospital, Maxwell AFB, Alabama.
Time and place of meeting: October 16, 1956, Palmer House, Chicago, Ill.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Treasurer: Dr. Norman Jesberg, 500 So. Lucas Ave., Los Angeles 17, Calif.
Otolaryngology: Dr. Russell M. Decker, 65 N. Madison Ave., Pasadena 1, Calif.
Ophthalmology: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 57, Calif.
Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

PUDGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
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CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE.

President: Dr. G. M. T. Hazen, 208 Canada Bldg., Saskatoon, Sask.
Secretary: Dr. G. Arnold Henry, 170 St. George St., Toronto, Ontario.
Meeting: Banff Springs Hotel, Banff, Canada, June 17-19, 1957.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Theodor Hunermann, Dusseldorf, Germany.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadephia 40, Pa., U. S. A.
Meeting: Sixth International Congress of Bronchoesophagology, Philadelphia, May 12-13, 1957.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting: Lima, Peru, 1957.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

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Vocal: Dr. Pedro Hernández Gonzalo.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

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Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

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Secretary of Exterior: Dr. Aldo G. Remorino.
Secretary Treasury. Prof. Dr. Antonio Carrascosa.
Pro-Secretary of the Interior: Prof. Dr. Carlos P. Mercandino.
Pro-Secretary of the Exterior: Prof. Dr. Jaime A. del Sel.
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SOCIEDAD COLUMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLUMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA.

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDIO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
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SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

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